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CLINICAL SURGICAL DIAGNOSIS

FOR

STUDENTS AND PRACTITIONERS

CLINICAL SURGICAL DIAGNOSIS

FOR

STUDENTS AND PRACTITIONERS

BY

F. DE QUERVAIN

Professor of Surgery and Director of the Surgical Clinic at the University of Basle

WITH 604 ILLUSTRATIONS AND 5 PLATES

2nd ENGLISH EDITION

Translated from the Fifth Edition by

J. SNOWMAN, M.D.

NEW YORK
WILLIAM WOOD AND COMPANY
MDCCCCXVII

JOHN BALE, SONS AND DANIELSSON, LTD.
OXFORD HOUSE
GREAT TITCHFIELD STREET, LONDON, W.

TRANSLATOR'S PREFACE TO THE SECOND ENGLISH EDITION.

THE exhaustion of the first English edition has coincided with the preparation of another new edition of the original work by Professor de Quervain. The present translation is accordingly made from the *fifth* edition of the original. Although it was practically complete before the outbreak of the War, many matters of interest from the standpoint of military surgery, were introduced while the work was going through the press. The last chapter is entirely devoted to problems connected with military surgery.

But the author's practical and graphic method of dealing with the surgery of civil life, still remains the outstanding feature of the book, and the present edition strives to exemplify this feature still further. Every chapter has undergone revision, but the sections on diseases of the stomach and duodenum have been rewritten entirely, on the basis of the most recent advances in clinical experience and radiological research.

There are ninety-two additional illustrations, and their introduction has permitted of an abbreviation of the text in some places. Plate III is a modification of the one which appeared in the previous edition, and it represents the result of the latest investigations of the Basle Anatomical Institute.

J. SNOWMAN.

50341

AUTHOR'S PREFACE TO THE ENGLISH EDITION.

THIS book, the English version of which affords me special pleasure, is the outcome of years of association with students, as a teacher, and with medical practitioners at consultations. It is intended to serve as a guide to the former in the vast field of surgical diagnosis which they are required to explore, and to recall to the latter, knowledge which has perhaps faded, while drawing their attention to new developments in diagnosis. To this end I have employed the methods of investigation which are available for the general practitioner, or at any rate which can easily be carried out for him. These methods comprise the bacteriological, serological, histological and radiographic researches, without which, nowadays, a reliable diagnosis cannot be obtained.

I adhere throughout to the plan of starting with the symptoms which caused the patient to seek medical advice, and not to the method of deducing symptoms from an already made diagnosis.

If the appropriate questions have been correctly put, the problem will in every case be narrowed down more and more, until, ultimately, the student arrives at a definite diagnosis, either as the result of positive symptoms or by a process of gradual exclusion. After long training in this method, the student may be permitted to diagnose a case as a whole, without first considering each symptom separately. One thing, however, must be borne in mind. Important as it is to have an accurate diagnosis, our endeavours to obtain one dare not entail injury to the patient, nor involve the loss of the most favourable moment for a successful operation, while refinements of diagnosis are being investigated. A diagnosis is not to be made merely for its own sake, but as a means to a cure.

This book represents, above all, the fruit of my own experience, and the illustrations are, with a few exceptions, derived from my own observations. The contents may, in consequence, be necessarily incomplete in certain particulars, but I trust that this is compensated for by vividness of description.

F. DE QUERVAIN.

Basle, February, 1913.

CONTENTS.

PART I.

SURGICAL DISEASES OF THE HEAD.

PAGE

I.—FRACTURES OF THE SKULL 1

1.—Direct Symptoms 2

2.—Indirect Symptoms 4

II.—INJURIES OF THE BRAIN 8

1.—Concussion of the Brain 9

2.—Contusion of the Brain 10

3.—Cerebral Pressure following Hæmorrhage 12

III.—ABSCESS OF BRAIN, HÆMORRHAGIC PACHYMENINGITIS, CYST OF BRAIN, TUMOUR OF BRAIN 18

1.—Abscess of Brain 19

2.—Hæmorrhagic Pachymenigitis 21

3.—Traumatic Cysts of the Brain 22

4.—New Growths and Tumours of Granulation Tissue ... 23

IV.—THE CEREBRAL COMPLICATIONS OF SUPPURATIVE OTITIS MEDIA 27

V.—THE PROBLEM OF EPILEPSY 37

VI.—SOME REMARKS ON CEREBRAL LOCALIZATION AND FOCAL DIAGNOSIS 41

VII.—THE SURGERY OF EXOPHTHALMOS 53

VIII.—ACUTE INFLAMMATORY PROCESSES ON THE SKULL ... 55

IX.—SWELLINGS OF THE HEAD 55

A.—Congenital Swellings of the Head 55

B.—Acquired Swellings of the Head 59

1.—Innocent acquired Swellings 59

2.—Malignant Tumours of the Head and Chronic Inflammatory Swellings 60

X.—ACUTE INFLAMMATORY DISEASES OF THE FACE ... 63

XI.—TUMOURS AND ULCERS OF THE FACE 67

A.—Tumours with Overlying Skin unbroken 67

B.—Ulcerative Processes 69

XII.—INJURIES OF THE JAW 80

XIII.—LOCKJAW 82

XIV.—INFLAMMATORY DISEASES OF THE JAW 86

PAGE
XV.—TUMOURS OF THE UPPER JAW 90
XVI.—TUMOURS OF THE LOWER JAW 93
XVII.—ACUTE INFLAMMATORY DISEASES OF THE ORAL CAVITY 96
XVIII.—TUMOURS AND ULCERS IN THE MOUTH, PHARYNX AND NOSE CAVITIES 99
A.—Non-ulcerating Growths 100
1.—The Mucous Membrane of the Lips and Cheeks 100
2.—The Floor of the Mouth 100
3.—The Gums 102
4.—Palate, Tonsillar Region, Base of Tongue 103
5.—Pharynx 104
6.—Nasal Cavity 106
B.—Ulceration Processes 107
1.—The Mucous Membrane of the Lips and Cheeks 107
2.—The Floor of the Mouth 108
3.—The Gums 108
4.—The Tonsillar Region 109
5.—Hard and Soft Palate 111
6.—Pharyngeal Wall 111
7.—Nasal Cavity 111
XIX.—CHRONIC DISEASES OF THE TONGUE 112
1.—Non-ulcerated Tumours and Swellings 113
2.—Ulcerative Diseases of the Tongue 115

PART II.

SURGICAL DISEASES OF THE NECK.

XX.—SURGICAL DISEASES OF THE RESPIRATORY TRACT (BRONCHI, LARYNX AND TRACHEA) 118
A.—Acute Diseases 119
1.—Inflammatory Processes in the Pharynx and Larynx 119
2.—Pure Circulatory Disturbances 122
3.—Injuries 123
4.—Foreign Bodies in the Air Passages 123
B.—Chronic Diseases 125
XXI.—DIFFICULTY IN SWALLOWING 130
A.—Disturbances of the Mechanism of Swallowing in the Mouth and Throat 130
B.—Disturbances of the Mechanism of Deglutition in the Region of the Œsophagus 132

PAGE
XXII.—ABSCESSES OF THE NECK 139
A.—Acute Inflammatory Processes 139
1.—The Sub-mental Region 140
2.—The Sub-maxillary Region 140
3.—The Side of the Neck 141
4.—Supra-clavicular Region 141
5.—Anterior Triangle of the Neck 141
6.—Posterior Cervical Region 142
B.—Chronic Abscesses 143
XXIII.—SINUSES IN THE NECK 145
XXIV.—TUMOURS AND ALLIED SWELLINGS OF THE NECK ... 148
A.—The Anterior Triangle 149
1.—The External Appearances of Goitre 150
a.—Diffuse Goitre 150
b.—Circumscribed Goitre 155
2.—Position of the Goitre 156
3.—Complications of Goitre: Hæmorrhage, Inflammation and Malignant Degeneration 159
a.—Hæmorrhage 159
b.—Inflammation 160
c.—Malignant Degeneration 162
B.—Tumours of the Side of the Neck and its Vicinity ... 164
1.—Enlargement of the Lymphatic Glands 164
2.—Tumours with Liquid Contents 169
3.—Solid Tumours of the Neck 176
a.—Sub-maxillary Region 176
b.—Parotid Region... 178
c.—Side of Neck 180
d.—Supra-clavicular Region 182
C.—Back of the Neck 184
XXV.—ABNORMAL POSTURES OF THE HEAD 186
A.—Painful Rigidity of the Neck 186
1.—Symmetrical Forms 187
a.—Rigidity with Sudden Onset 187
b.—Rigidity with Gradual Onset 193
2.—Asymmetrical Forms 193
B.—Painless Rigidity of Neck... 196

PART III.

SURGICAL DISEASES OF THE THORAX.

XXVI.—FRACTURES OF THE BONES OF THE THORAX 199
XXVII.—INJURIES OF THE LUNG 201
XXVIII.—INJURIES OF THE HEART 204
XXIX.—THE SURGERY OF INFLAMMATORY DISEASES OF THE LUNG 207
A.—Empyema, Abscess of the Lung, Gangrene of the Lung 208
B.—Bronchiectasis 211
C.—Actinomycosis 212

PAGE
XXX.—TUMOURS AND ALLIED GROWTHS WITHIN THE THORAX 212
1.—Mediastinal Tumours 212
2.—Tumours of the Lung 221
XXXI.—SWELLINGS AND TUMOURS OF THE THORAX 222
A.—Primary Disease within the Thorax 222
B.—Primary Disease of the Thoracic Wall 223
1.—Acute Diseases 224
2.—Chronic Diseases 224
a.—Chronic Inflammatory Processes 225
b.—Tumours 229
XXXII.—INFLAMMATORY DISEASES OF THE BREAST 233
1.—Acute Inflammations 235
2.—Chronic Inflammations 236
XXXIII.—TUMOURS AND ALLIED STRUCTURES IN THE BREAST 238
A.—Multiple Tumours 239
B.—Single Tumours 239
1.—Small and Medium-sized Tumours 239
2.—Large Tumours 247

PART IV.

SURGICAL DISEASES OF THE ABDOMINAL AND PELVIC VISCERA.

XXXIV.—DISPLACEMENTS OF THE ABDOMINAL VISCERA 251
XXXV.—ABDOMINAL INJURIES 260
A.—Injuries without an Open Wound 260
1.—Gastro-intestinal Canal 261
2.—The Spleen 263
3.—Liver and Bile Ducts 263
4.—The Kidneys 265
5.—The Bladder 266
B.—Injuries to the Abdomen with Open Wounds 268
1.—Gunshot Wounds 268
2.—Stabs and Incised Wounds 269
XXXVI.—ACUTE INFLAMMATION WITHIN THE ABDOMINAL CAVITY 270
A.—Abdominal Pain without Perceptible Changes 278
B.—Diffuse Peritonitis without Localization 280
C.—Localized Peritonitis 282
1.—Epigastrium 282
2.—Right Hypochondrium 282
3.—Left Hypochondrium 282
4.—Lumbar Regions 282
5.—Lower Abdominal Region 283
6.—True Pelvis 292

PAGE

XXXVII.—SUB-PHRENIC ABSCESS 293

1.—Sub-phrenic Abscess without Pleural Effusion 294

2.—Sub-phrenic Abscess with Pleural Effusion 297

XXXVIII.—TUBERCULAR PERITONITIS 298

XXXIX.—DIAGNOSIS OF ABDOMINAL SWELLINGS IN GENERAL ... 304

XL.—SURGICAL DISEASES OF THE STOMACH AND DUODENUM 309

A.—Foreign Bodies in the Stomach 320

B.—Displacements of the Stomach 320

C.—Gastric and Duodenal Ulcer 321

1.—Position of Ulcer 321

2.—Diagnosis of Secondary Innocent Changes 324

3.—Diagnosis of Malignant Degeneration of Ulcer 325

D.—Primary Cancer of the Stomach and Duodenum 325

1.—Cancer of the Cardiac End of Stomach 327

2.—Cancer of the Body of Stomach 327

3.—Cancer of Pylorus 328

XLI.—ACUTE PERFORATION OF GASTRIC AND DUODENAL ULCER 329

XLII.—SURGICAL DISEASES OF THE BILIARY PASSAGES 332

1.—Gall-stone Colic 333

2.—Acute Cholecystitis 335

3.—Gangrenous Cholecystitis 338

4.—Obstruction of the Common Bile-duct 340

5.—Hydrops of the Gall-bladder, Chronic Empyema 343

6.—Acute Cholangitis 345

XLIII.—TUMOURS OF THE LIVER 345

XLIV.—SURGERY OF THE PANCREAS 348

1.—Acute Pancreatitis and Pancreatic Hæmorrhage... ... 348

2.—Chronic Pancreatitis: Cancer of the Head of the Pancreas: Pancreatic Calculus 350

3.—Pancreatic Tumours and Cysts 350

XLV.—SURGERY OF THE SPLEEN 351

1.—Abscess of the Spleen 351

2.—Splenic Hypertrophy... 352

3.—Tumours of the Spleen 352

XLVI.—ACUTE APPENDICITIS 353

XLVII.—COLITIS, SO-CALLED CHRONIC APPENDICITIS AND FUNCTIONAL DISTURBANCES OF THE LARGE INTESTINE 360

A.—Colitis with Definite Anatomical Changes... 361

B.—Functional Derangements of the Large Intestine without Typical Anatomical Changes 363

PAGE
XLVIII.—INTESTINAL OBSTRUCTION 368
1.—STENOSIS OF GRADUAL DEVELOPMENT (CHRONIC INTESTINAL OBSTRUCTION) 368
a.—Symptoms 368
b.—The Position of the Stenosis 372
c.—Form and Cause of the Stenosis 372
2.—ACUTE INTESTINAL OBSTRUCTION 380
a.—Symptoms 380
b.—The Position of the Obstruction 381
c.—The General Varieties of Acute Obstruction 384
d.—Causes of Acute Intestinal Obstruction 385
1.—Obstruction due to Bands and Kinks 385
2.—Obstruction by Gall-stones 386
3.—Intussusception 387
4.—Volvulus 388
5.—Strangulation of Internal Herniæ 389
6.—Spastic Obstruction 390
XLIX.—TUMOURS AND SWELLINGS IN THE ABDOMINAL PARIETES 391
1.—THE UPPER ABDOMINAL REGION 392
2.—THE UMBILICAL REGION 394
3.—INGUINAL REGION 397
4.—THE LUMBAR REGION 398
5.—SWELLINGS AND TUMOURS IN ATYPICAL POSITIONS 399
L.—ABDOMINAL SINUSES 401
LI.—EXTERNAL INGUINAL HERNIA 403
1.—DIAGNOSIS IN THE ABSENCE OF HERNIAL SWELLING 404
2.—DIAGNOSIS OF INGUINAL HERNIAL SWELLINGS 406
3.—DIAGNOSIS OF LABIAL AND SCROTAL HERNIA 411
LII.—INTERNAL OR DIRECT INGUINAL HERNIA 413
LIII.—FEMORAL HERNIA 416
LIV.—TRAUMATIC HERNIÆ 421
LV.—STRANGULATED HERNIA 422
1.—IS THE CASE REALLY ONE OF HERNIA? 423
2.—IS THE HERNIA STRANGULATED? 425
3.—WHAT DOES THE HERNIA CONTAIN? 427
4.—WHERE IS THE STRANGULATION SITUATED? 427
5.—WHAT IS THE CONDITION OF THE STRANGULATED GUT? ... 428
6.—THE QUESTIONS WHICH MAY ARISE DURING OPERATION ... 428
7.—QUESTIONS WHICH ARISE AFTER REDUCTION BY THE OPEN OR BLOODLESS METHOD 430
LVI.—DIFFICULTIES OF DEFÆCATION 431
LVII.—INJURIES OF THE RECTUM 437
LVIII.—TUMOURS AND SWELLINGS OF THE SCROTUM 439
1.—SWELLINGS OF THE SCROTUM 439
a.—Acute Swellings 439
b.—Chronic Swellings 440
2.—SWELLINGS OF THE SCROTAL CONTENTS 441
a.—Tumours of the Spermatic Cord 441
b.—Acute Swellings of the Testicle and Epididymis 442
c.—Chronic Swellings of the Testicle and Epididymis 445
1.—Swellings of the Epididymis 445
2.—Swellings between the Testicle and Epididymis 446
3.—Swellings of the Testicle 447

PAGE

LIX.—FISTULÆ IN PERINEAL REGION 450
1.—Dermoid Fistulæ 450
2.—Fistulæ in Connection with Bone 451
3.—Fistulæ of the Rectum and Anus 451
4.—Urinary Fistulæ 454

LX.—GENERAL REMARKS ON THE SURGICAL DISEASES OF THE URINARY ORGANS 455
I.—Disturbances of Micturition 456
a.—Painful Micturition 456
b.—Difficult Micturition 457
1.—Disturbances of the Mechanism of Micturition 457
2.—Obstruction of the Urethra 457
c.—Deficient Closure of the Bladder 459
d.—Vesical Tenesmus 460
II.—Abnormal Composition of the Urine 461
a.—Admixture with Pus 461
b.—Admixture with Blood 463
c.—Admixture with Inorganic Deposits or Concretions (urinary gravel) 464
III.—Local Symptoms 465

LXI.—INFLAMMATION IN THE NEIGHBOURHOOD OF THE KIDNEY 470

LXII.—MOVABLE KIDNEY 473

LXIII.—HYDRONEPHROSIS AND ITS CONSEQUENCES 476

LXIV.—IDIOPATHIC SUPPURATION IN THE RENAL PELVIS AND KIDNEY 478

LXV.—STONE IN THE KIDNEY AND URETER 482
A.—Primary Stone in the Kidney 482
1.—Non-infected Stone 482
2.—Infected Stone 483
B.—Secondary Stone in the Kidney 483

LXVI.—TUMOURS OF THE KIDNEY 485

LXVII.—TUBERCULOSIS OF THE URINARY PASSAGES 488

LXVIII.—STONE IN THE BLADDER 491

LXIX.—CYSTITIS 494

LXX.—TUMOURS OF THE BLADDER 496
1.—Tumours of the Mucous Membrane of the Bladder ... 496
2.—Tumours in the Muscular Coat of the Bladder 497

LXXI.—HYPERTROPHY, TUMOURS, AND ABSCESS OF THE PROSTATE 498
1.—Hypertrophy and Tumours 498
2.—Inflammatory Processes 501

LXXII.—INJURIES OF THE URETHRA 502

LXXIII.—SURGICAL DISEASES OF THE PENIS 505
1.—Subcutaneous Growths 505
2.—Ulcerative Changes 506

PART V.

SURGICAL DISEASES OF THE PELVIS AND SPINAL COLUMN.

PAGE

LXXIV.—TUMOURS OF THE PELVIS 509

LXXV.—CONGENITAL ABNORMALITIES OF THE SPINAL COLUMN 513

LXXVI.—LUMBAGO 518

LXXVII.—INJURIES OF THE SPINAL COLUMN 520

- I.—METHOD OF EXAMINATION 521
- II.—DIAGNOSIS OF THE NATURE, DEGREE, AND POSITION OF THE INJURY 525
  - A.—The Degree and the Nature of the Spinal Cord Injury 525
  - B.—The Position of the Spinal Cord Injury (the Diagnosis of the level of the Lesion) 526
  - C.—Relations between the Injury to the Cord and the Vertebræ ... 531
  - D.—The Form of the Spinal Injury 533
    - 1.—Fractures of the Spinous and Transverse Processes... ... 535
    - 2.—Fracture of the Vertebral Arch 536
    - 3.—Compression Fracture 537
    - 4.—Complete Dislocation 538

LXXVIII.—THE SURGERY OF NON-TRAUMATIC DISEASES OF THE SPINAL CORD... 540

- 1.—IS A TUMOUR ACTUALLY PRESENT? 541
- 2.—WHAT IS THE NATURE OF THE TUMOUR? 542
- 3.—AT WHICH LEVEL IS THE TUMOUR SITUATED? 543

LXXIX.—INFLAMMATORY DISEASES OF THE SPINAL COLUMN 544

- A.—TUBERCULAR CARIES 544
  - 1.—Tubercular Caries without definite Curvature, and without a Burrowing Abscess 544
  - 2.—Spinal Caries with Burrowing Abscess 549
  - 3.—Spinal Caries with Curvature 552
  - 4.—Spinal Caries with Cord Symptoms 553
- B.—NON-TUBERCULAR INFLAMMATION OF THE SPINAL COLUMN ... 553

LXXX.—SPINAL CURVATURES 555

- 1.—ANTERO-POSTERIOR CURVATURES 559
- 2.—LATERAL CURVATURES 559

PART VI.

SURGICAL DISEASES OF THE UPPER EXTREMITY.

PAGE
LXXXI.—FRACTURES AND DISLOCATIONS OF THE CLAVICLE ... 568
LXXXII.—FRACTURES AND DISLOCATIONS ABOUT THE SHOULDER-JOINT ... 572
A.—DISLOCATIONS ... 573
B.—FRACTURES ... 574
LXXXIII.—INFLAMMATORY PROCESSES ABOUT THE SHOULDER-JOINT ... 581
A.—DISTINCTION BETWEEN BURSAL AND JOINT DISEASE ... 581
B.—DIAGNOSIS OF THE VARIOUS FORMS OF BURSITIS AND ARTHRITIS OF THE SHOULDER ... 584
1.—Bursitis ... 584
2.—Arthritis of the Shoulder ... 584
C.—PRIMARY DISEASES OF THE BONE ... 587
LXXXIV.—INJURIES ABOUT THE ELBOW-JOINT ... 587
A.—INSPECTION ... 588
B.—EXAMINATION OF ELBOW MOVEMENTS ... 588
C.—PALPATION ... 594
D.—RÖNTGEN RAY EXAMINATION... 600
LXXXV.—INFLAMMATORY PROCESSES ABOUT THE ELBOW ... 603
1.—ACUTE INFLAMMATORY PROCESSES ... 603
2.—CHRONIC INFLAMMATORY PROCESSES ... 604
LXXXVI.—TUMOURS AND ALLIED SWELLINGS ON THE UPPER ARM AND FOREARM ... 607
LXXXVII.—INJURIES OF THE WRIST AND HAND ... 612
1—RADIUS AND ULNA ... 612
2.—WRIST-JOINT ... 615
3.—METACARPUS AND FINGERS ... 621
LXXXVIII.—INFLAMMATORY PROCESSES ABOUT THE WRIST ... 623
1.—ACUTE INFLAMMATIONS... 623
2.—CHRONIC INFLAMMATORY PROCESSES ... 625
LXXXIX.—ABNORMAL POSITIONS AND POSTURES OF THE HAND AND FINGERS ... 629
A.—RESULTS OF INJURIES TO NERVES ... 629
B.—ABNORMAL POSTURES OF THE WRIST-JOINT ... 632
C.—ANOMALIES IN THE POSTURE OF THE FINGERS ... 635
XC.—TUMOURS OF THE HAND AND FINGERS ... 636
A.—INNOCENT TUMOURS ... 636
B.—MALIGNANT TUMOURS ... 639
XCI.—ACUTE INFLAMMATORY PROCESSES OF THE HAND AND FINGERS ... 640
A.—INFLAMMATORY PROCESSES OF THE FINGERS ... 641
B.—ACUTE INFLAMMATORY PROCESSES OF THE HAND ... 643
XCII.—CHRONIC INFLAMMATION OF THE HAND AND FINGERS 644
1.—THE SKIN ... 644
2.—THE TENDON-SHEATHS ... 646
3.—THE BONES ... 647

PART VII.

SURGICAL DISEASES OF THE LOWER EXTREMITY.

PAGE
XCIII.—DISLOCATIONS AND FRACTURES OF THE HIP 650
  A.—Method of Examination 651
  B.—Diagnosis of the Various Forms of Injury 655
    1.—Dislocations 655
    2.—Contusions, Sprains, Fractures 661
XCIV.—NON-TRAUMATIC DEFORMITIES AT THE HIP-JOINT (CONGENITAL DISLOCATION OF THE HIP AND COXA VARA) 671
  A.—Congenital Dislocation of the Hip 671
  B.—Coxa Vara 679
XCV.—ACUTE INFLAMMATORY DISEASES OF THE HIP-JOINT 684
XCVI.—CHRONIC INFLAMMATORY DISEASES OF THE HIP ... 686
  A.—Tubercular Hip Disease 686
  B.—Non-tubercular Chronic Hip Disease 694
XCVII.—SWELLINGS AND TUMOURS OF THE THIGH 696
  A.—Swellings of the Soft Tissues 698
  B.—Swellings of the Bone 700
    1.—Osteoma and Chondroma 700
    2.—Sarcomata and Allied Tumours 700
      a.—Tumours of the Epiphysis 701
      b.—Tumours of the Diaphysis 705
XCVIII.—INJURIES WITHIN THE VICINITY OF THE KNEE-JOINT 708
XCIX.—ACUTE INFLAMMATORY DISEASES OF KNEE-JOINT ... 719
C.—CHRONIC DISEASES OF THE KNEE 721
  1.—Chronic Articular Effusion 721
  2.—Fungating Inflammation of the Knee-joint 726
  3.—Rigidity 728
CI.—TUMOURS AND ALLIED STRUCTURES ABOUT KNEE-JOINT 729
CII.—SCIATICA AND OTHER PAINFUL DISEASES OF THE LOWER LIMBS 731
CIII.—ULCERS OF THE LEG 735
CIV.—SWELLINGS AND TUMOURS OF THE LEG 741
  A.—Acute Diseases 743
  B.—Chronic Inflammatory Processes and Tumours 743
    1.—Diffuse Inflammatory Processes 746
    2.—Circumscribed Swellings 748
CV.—INJURIES ABOUT THE ANKLE-JOINT 752
  1.—Injuries without Deformity 752
  2.—Injuries with Deformity 759
CVI.—FRACTURE OF THE OS CALCIS 763

PAGE

CVII.—INJURIES TO THE FOOT, IN FRONT OF THE ANKLE-JOINT ... 768

CVIII.—INFLAMMATORY DISEASES OF THE FOOT ... 771
  A.—Tarsus ... 771
    1.—Acute Diseases ... 771
    2.—Chronic Inflammations ... 772
  B.—Metatarsus and Toes ... 775

CIX.—DEFORMITIES OF THE FOOT ... 776
  1.—Flat Foot ... 776
  2.—Talipes, Pes equinus, Pes cavus, Pes calcaneus ... 782
  3.—Deformities of the Toes ... 787

CX.—TUMOURS AND ULCERS OF THE FOOT ... 787
  1.—Tumours ... 787
  2.—Ulcers ... 789

CXI.—SOME GENERAL DIAGNOSTIC PROBLEMS IN CONNECTION WITH THE MILITARY SURGERY OF THE LIMBS ... 791
  1.—Evidence of Injury to Bones ... 791
  2.—Evidence of Injury to Joints ... 792
  3.—Diagnosis of Injuries of Large Blood Vessels ... 793
  4.—Diagnosis of Injuries of Nerves ... 794
  5.—Evidence of Presence of Foreign Bodies ... 796
  6.—Diagnosis of Infection of the Limbs after War Injuries 803
    a.—Ordinary Suppuration ... 804
    b.—Erysipelas ... 804
    c.—Infections associated with gas formation ... 805
    d.—Tetanus ... 806

Index ... 807

PART I.

SURGICAL DISEASES OF THE HEAD.

CHAPTER I.

FRACTURES OF THE SKULL.

When Dieffenbach summarized his views on trephining, in his classical work, some sixty years ago, before the question was definitely settled, he wrote as follows :—

> "Up to quite recently it was the first urgent duty of the surgeon, immediately after the injury, to make a large crucial incision, and to search for fissures or fractures. Occasionally I found on my arrival that the hairy portion of the head had been ploughed up by incisions, backwards and forwards, crucial and transverse. This was considered to be indispensable in all head injuries. A surgeon who had omitted to make the crucial incision would have incurred the same responsibility as one who failed to open the window in endeavouring to rescue a victim of coal-gas suffocation."

At that time there was no "cerebral diagnosis," but, nevertheless, the surgeon's need for a diagnosis had to find some expression. To leave an injury to Nature, without knowing whether or not a fissure was present, was considered to be an unworthy and culpable piece of surgical inactivity, because the real danger of a fractured skull had not yet been clearly ascertained. Philosophical speculation took the place of observation, and therefore surgery for a long time was guilty of one of its greatest sins, in breaking the law *primum nil nocere.* We smile at this kind of diagnosis, but posterity will look upon some of our exploratory procedures, involving flaps of half the skull, much in the same way as we regard those meaningless "crucial and transverse incisions." The maxim "nil nocere" must be observed, not only in treatment, but also in examination.

The term "fracture of the skull" has always conveyed to the lay mind a very painful impression, but we have learnt, since the days when trephining was epidemic, that it is not the fracture of the skull, nor even its splintering, which constitutes the danger, but the *injury* *to the brain.* It is all-important to recognize this, because this alone can justify or compel interference. The study of *injuries to the skull* is of great assistance in this respect, and we must endeavour to gain a precise conception of them—not by pouring pigment on the exposed skull after the manner of the older surgeons, but by a careful consideration of all clinical symptoms.

(1) DIRECT SYMPTOMS.

The anatomical structure of the skull frequently conceals the usual symptoms of fracture, but the main indications are always present. The symptoms may be summarized as : (1) Pain at the seat of fracture ; (2) displacement of fragments ; (3) unnatural mobility, and (4) hæmorrhage.

(1) The **pain** in fractured skull may be evident in two ways. Pain on *direct* pressure signifies that great care is demanded. A limited area of tenderness on pressure is of no importance, because every bruise is eventually painful. But a definite pain persisting along one line, for several days, should, at any rate, suggest the probability of a complete fissure. Pain may also be elicited at the seat of fracture, by *indirect* pressure, either by compressing the whole skull, or by applying pressure to one of the large fragments. This sign may be important in the case of fissures ; but in the case of a comminuted fracture, the unnatural mobility will already have established the diagnosis.

(2) **Displacement** and **unnatural mobility** only occur in comminuted fractures. Displacement manifests itself in the form of a depression—*depressed fracture*—and unnatural mobility by resilience on pressure. These two symptoms do not usually occur together, because the fragments become fixed in a depressed fracture, and the depression remains; whereas, if the fragments are movable, the intracranial pressure tends to replace them, and the original depression is spontaneously raised. It is quite easy to recognize these symptoms, and either of them is conclusive proof of a comminuted fracture of the roof of the skull. There is only one possibility of error, which has misled even experienced surgeons ; whenever any extensive hæmorrhage occurs under the aponeurosis, or especially under the periosteum, the effused blood feels at the side like a gently sloping, thick mound with a sharp edge, corresponding with the blood-soaked tissues ; whereas at the centre, where the blood collects in a larger hollow space, the consistence is softer, and to the examining finger it may appear as a depression. It makes one think first of a depressed fracture. But if gradually increasing pressure causes the disappearance of the mound-like swelling, at any one spot, it is

obviously a large contusion—a cephalhæmatoma—and not a depressed fracture.

The extensive hæmatomata which occur in alcoholics through vascular degeneration easily impart to the finger the sensation of having penetrated within the skull.

A young female, well known as an alcoholic of an advanced type, was once admitted to hospital under my care. Her husband had beaten her about the head with the leg of a chair, so that it resembled a globular mass without the sign of a human feature. The scalp was one crackling area, and I was convinced that a severe fracture was present beneath it. But the withdrawal of a few ounces of blood by means of a puncture rapidly restored things to normal, and I found nothing subsequently to give the least support to a diagnosis of fracture. The only " cerebral symptom " she ever had was the noise with which she came to the Hospital.

It is important not to be deceived by congenital peculiarities in the shape of the skull, such as a deep hollow in the occipital bone, or by the presence of Wormian bones, by syphilitic ulceration, or by depressions due to former injuries.

There are some people to whom accidents are doled out by Fate with exceptional abundance. Thus, I have seen a man who sustained a fractured skull twice within a year, in attempts, as he said, to "preserve the peace" in a public-house. He at once explained that a depression found on examination dated back to a similar incident, ten years before.

(3) **Hæmorrhage** is a much more constant, and therefore a more significant symptom. It may indeed be the only evidence of a fractured base. After any head injury, immediate inquiry should be made for bleeding from the ear, mouth or nose.

In *bleeding from the ear* it is necessary to be sure that the blood does not originate from the auditory canal. If there be but little blood in the canal, it may have been caused merely by a ruptured tympanic membrane, which can occur without a fractured base. More profuse or persistent bleeding, however, points with great probability to a bone injury, which indicates a fractured base, unless the external auditory passage has itself been severely wounded.

The auditory canal has in rare cases been injured by the pressure of an anterior displacement of the lower jaw, consequent upon a blow.

Hæmorrhage from the *nose* and *mouth* is only significant when the injury has not directly involved the face. If such injury can be excluded, it points to a fissure in the ethmoid, sphenoid or anterior part of the basilar process of the occipital bone. Exceptionally, the blood might come from the Eustachian tube.

Diagnostic importance attaches to *subcutaneous effusions of blood*, as well as to its immediate escape externally. Their situation and their mode of onset are both significant. They usually appear in the region of the orbit and mastoid process. They do not appear directly after the injury, as do effusions from contusion. It requires some few hours for the blood to reach the superficial tissues; occasionally it takes a few days. We must guard against diagnosing every " black eye" as a fractured base. The possibility of the face having been involved in the injury must always be considered; the patient may have fallen on his face in addition to the primary accident. But in the absence of such a cause, we may definitely conclude that there is an orbital fracture, generally on the roof, when close observation reveals a gradual protrusion of the eyeball, a bluish discoloration of the eyelids and hæmorrhagic infiltration of the eyelids—signs all pointing to bleeding within the fatty and cellular tissue of the orbit. If an effusion of blood appears behind the ear a day or two after the injury, we may conclude that the fracture is either in the middle or posterior cranial fossa. If the patient has not come under observation until the second or third day, information as to the onset of the ecchymoses, at any rate the most striking ones in the lid and conjunctivæ, must be obtained from the friends.

A mistake might easily have arisen in the following circumstances. A young man, who had been run over by a cart, was brought into hospital with subconjunctival ecchymoses. A superficial examination suggested a fractured base, but he had numerous punctiform hæmorrhages over his face, neck, and upper portion of the chest. It was evidently a case of *congestive hæmorrhages* due to compression of the thorax. Some slight cerebral symptoms followed, which indicated that the effects of the congestion had also been felt within the skull.

(4) After cessation of the hæmorrhage, clear fluid may escape from the nose or ear. If this liquid contain little albumin and much sodium chloride it is cerebrospinal fluid, and this affords an additional aid to diagnosis, which is still further confirmed by the escape of *brain substance*. Fortunately, it is not very often that this latter is mistaken for " pus " by a zealous practitioner.

(2) INDIRECT SYMPTOMS.

The direct symptoms of fracture are frequently complicated by **secondary injuries**, which require careful attention from the point of view of diagnosis and of prognosis. These complications concern the **cranial nerves**, and in the section treating of cerebral localization we shall discuss the differential diagnosis from central palsies.

Facial paralysis is the commonest of these lesions.

The nerves to the ocular muscles are sometimes involved, more especially the sixth nerve, which may indeed be affected on both sides. The third and fourth nerves are less frequently involved, and injury to the other cranial nerves is quite exceptional.

The nature of the nerve lesion can be inferred from the time of onset of these paralyses and from their severity. If the paralysis supervenes immediately on the injury and is complete, it is obvious that the nerve must be crushed or torn at its point of exit from the pons or medulla. If its onset is delayed for some hours it suggests compression by hæmorrhage ; if delayed for some days the possibility of infective neuritis may be entertained, in the case of a complicated fracture. In the two latter conditions the paralysis gradually increases, but it will probably remain incomplete.

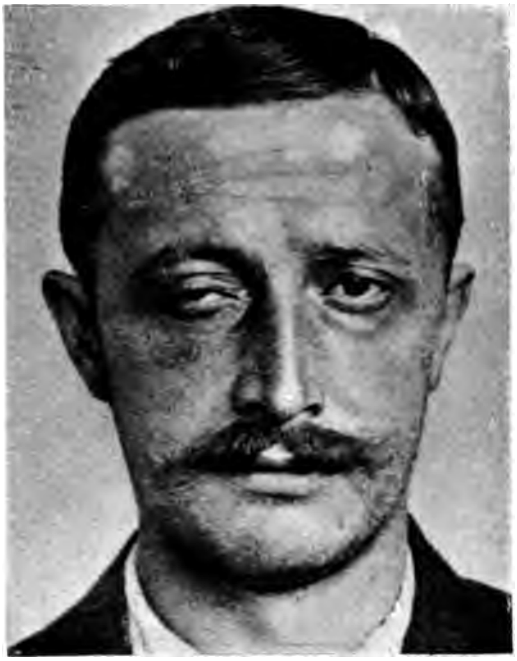


FIG. 1.—Paralysis of third nerve on right side in a case of fractured base of skull (ptosis and paralysis of internal rectus).

The following case illustrates the care which must be exercised in the diagnosis of these secondary injuries :—

A workman sustained a severe blow on the top of the head, against a stone floor. A gradually increasing oculomotor paralysis of the left side appeared on the second day. Even if the patient had not been insured against accidents, one would have thought of the connection between the injury and the paralysis. But subsequent detailed examination showed that, not only were all the external muscles of the left eye paralysed, but there was loss of pupil reflexes in both eyes. The site of the lesion must, therefore, have been in the region of the nucleus. Inquiries made of the ophthalmic surgeon, whom the patient had consulted a year previously for a slight accident, elicited the fact that already at that time he had no pupil reflexes. But no evidence of syphilis could then be ascertained. The left-sided ocular paralysis improved under mercurial treatment, which was undertaken by way of experiment, but right-sided paralytic symptoms developed subsequently. The injury, therefore, came at an unpropitious moment, but it evidently was not the sole cause of the trouble.

I saw the patient again a few years later, and on this occasion also for a slight injury to the skull. He had constant double vision, and Wassermann's reaction was positive.

Injuries of the optic nerves frequently occur with a fractured base. As a rule, optic atrophy appears after a little time. Sometimes the nerve is only crushed, so that the blindness is not complete; but the prospect of recovery is only small, because the optic nerve does not undergo regeneration like a peripheral nerve. Sometimes its blood supply sustains an injury, so that ophthalmoscopic examination reveals the appearance of obstruction of the central artery.

Deafness results, either from the not infrequent damage to the *auditory nerve itself*, or from involvement of the *labyrinth*, or the *vestibular nerve.* The symptoms of the latter are noises in the ear, giddiness, and nausea; but the latter must not be confused with the nausea caused by cerebral pressure. Labyrinthine concussion can occur independently of bone injury, so that a diagnosis of fractured base cannot be made from this condition alone.

After recognizing the presence of a fracture of the skull by means of the previously described symptoms, it is our duty to determine its **course and direction.**

Assistance is afforded for this purpose by the visible objective signs, and by the position of application of the force, if this is ascertained from the history or by the site of injury on the soft tissues. The skull is *bent* at the point whereon the blow is dealt; if the force is limited in area the bone breaks concentrically and a *perforated fracture* is produced. Thence, cracks radiate towards the opposite pole, *i.e.*, generally towards the base. These constitute the *split fractures.* Sometimes these cracks start at the base of the skull, because this is its weakest section, and run towards the site of the blow; but occasionally they fail to reach it. This explains why so many fissures are limited to the base. *Comminuted fractures* are always surrounded by fissures, produced by bending and splitting. Thoma simplifies this scheme still more, and includes both forms under the term "*deformity fractures*" (*Deformationsbrüche*), due to the abnormal reaction of the bone to traction and pressure.

Much unnecessary discussion has, in the past, been devoted to the question of separate fracture of the internal and external tables of the skull. It is, of course, true that one table may be fractured by itself, in which case fractures of the inner table are more extensive than those of the outer. This, however, is not a matter of diagnostic or therapeutic importance, and we may be quite satisfied if our diagnoses of penetrating fractures are not too often in error.

So-called *contrecoup fractures* are full of interest, and have given rise to much discussion, but they must be clearly distinguished from the split fissures of the base, just described. The existence of these fractures is doubted by some authorities.

As the skull is not a rigid structure, but of an elastic nature and subject to change in shape, a portion of the actual force of every blow which it sustains is received by the brain, and is transmitted through it to the opposite pole. The force is checked at this spot, and the brain sustains a "contrecoup," causing more or less contusion—a contrecoup contusion. If the bone is very thin at this spot (orbital roof, possibly also the tegmen tympani) it may break under the force of the cranial impulse, or even be completely pressed out of the skull (contrecoup fracture). These fractures at the opposite pole, caused by the continuation of the force within the cerebral substance, are quite independent of the fractures at the base produced by bending and splitting, and which are due to direct damage to the skull cap. The accompanying figures (2 and 3) clearly illustrate these varieties of fracture. They represent a case which I had the opportunity of observing whilst acting as assistant.

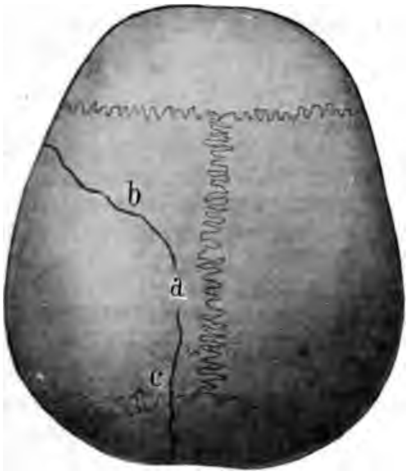


Fig. 2.—(After Kocher.) *a* = Site of application of blow; *b* and *c* = fissures produced.

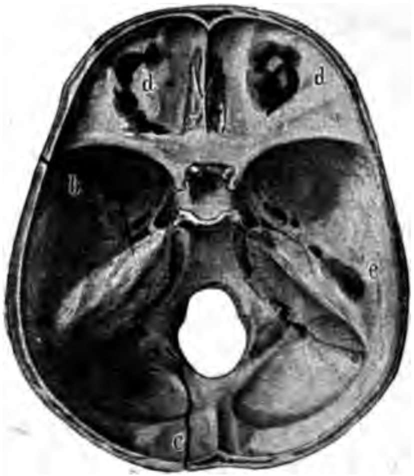


Fig. 3.—(After Kocher.) *b* and *c* = Basal portion of fissures; *dd* = separate contrecoup fractures of orbital roof; *e* = effusion on tegmen tympani.

Similarly the force exerted laterally by a projectile shot through the skull, from temple to temple, may result in pressing the orbital roof outwards, *i.e.*, into the orbit.

Attention to these points, and to the observations made on the patient, will facilitate an approximate diagnosis of the direction of the fissures, and particularly the fossa which they traverse. A confident diagnosis of contrecoup fracture of the orbital roof can only be made *post mortem*, because clinically we cannot exclude the possibility of its being caused by the primary injury.

Diagnosis by means of X-rays has not much practical value, in fractures of the skull, although in rare cases they may demonstrate fissures very distinctly. The observer must take care not to be deceived by sutures and arterial grooves.

CHAPTER II.

INJURIES OF THE BRAIN.

As we have already remarked, the treatment of fractured skull is determined by the injury to the brain, and not to the bone. For this reason our main attention must be devoted to it. Injuries to the brain may be divided into three classes: (1) *Concussion;* (2) *compression by hæmorrhage;* (3) *contusion.* It is useful to ascribe a special clinical picture to each variety; but in practice the differentiation is not very clear, and the different forms tend to coalesce. But individual features of each class can be recognized with more or less certainty, and separated from each other. An example will make this clear:—

A young stoker, who fell off his engine, was picked up somewhat stunned; he remounted, and after the stupefaction had passed off he became very excited. Vomiting, headache, and retardation of the pulse appeared in two hours, and after another couple of hours had passed he was completely unconscious. Examination showed that the left temporal bone was involved. On trephining, it was found that an extradural and an intradural hæmatoma existed under the fractured bone. A second trephining, the next day, revealed a right-sided intradural hæmorrhage. On the day after the injury, the temperature rose to 104° F., and the pulse became very rapid, so that the prognosis was obviously bad. Death took place on the third day.

This case permitted the formation of an accurate diagnosis step by step during life.

The stunning which came on immediately after the fall indicated *concussion.* The brief interval of freedom which preceded the severe symptoms of pulse retardation and unconsciousness pointed to an increasing *cerebral pressure* caused by the *hæmorrhage,* which was revealed at the operation. As the trephining which was done immediately, on the site of the injury, did not yield complete relief, it was obvious that there must be either another hæmatoma or a contusion of the brain or both together. The slow pulse becoming so rapid pointed to the probability of an increase in cerebral pressure through a second hæmatoma, which was found when the contrecoup area was trephined. The simultaneous rise of temperature led to the diagnosis of *cerebral contusion,* which was also verified at the *post-mortem.* There was no meningitis present, so that the pyrexia could not be attributed to this cause.

This is not the place to discuss the varying views which are held concerning cerebral pressure, and the numerous explanations which have been offered for so-called cerebral concussion. Fortunately, the careful clinical observation of the injured affords us sufficient indications for treatment, although we may not be able to explain physiologically every individual incident in the case.

Before we ascribe individual symptoms, especially vomiting, to a cerebral origin, we must assure ourselves that there is no other injury in addition to the fractured skull, as for instance an abdominal lesion. A young man, with a fractured skull, began to vomit on the second day, and this was naturally regarded as a cerebral symptom. Examination, however, showed that there was effusion in the abdomen, and laparotomy revealed a rupture in the hilus of the spleen. Finally it must be remembered that cerebral symptoms, such as vomiting and stupor, may supervene after any severe injury, especially fractured limbs. These symptoms are due to fat emboli, and they are associated with abundant fat in the urine, a marked acceleration of the pulse, and probably bloody sputum.

(1) CONCUSSION OF THE BRAIN.

We begin with the mildest degree of brain injury, the so-called **concussion of the brain.** It must be clearly understood that this term as usually applied is a *general term*, embracing all the milder forms of brain injury. A mild, rapidly evanescent cerebral pressure is also spoken of as concussion of the brain. "Concussion" is also a very comforting diagnosis when an indefinite contusion is sustained, especially on a part of the cortical area, whose function is unknown.

It is very convenient to have such a wholesale term, and the inadequacy of the diagnostic methods available, when an accident has just occurred, compels the practitioner to resort to this term, if he does not wish to acknowledge the impossibility of making an immediate diagnosis. But in truth, the term "concussion of the brain" is of most use to the journalist, who regards it as part of his professional duties to provide his readers with a ready-made diagnosis.

We will, for the present, put aside the cases due to pressure on the brain, or to brain contusion, and consider those which may be attributed to a transient functional disturbance, or, to be more precise, a vascular disturbance. These circulatory disorders have only two absolutely diagnostic signs; one is the fact that they begin immediately after the accident, and the other is their brief duration. The very definition of the term concussion demands that its onset should, as a *sine qua non*, follow forthwith upon the injury. Any interval before its appearance puts pure concussion out of the question. This diagnostic point is available at once; the other, the brief duration of the symptoms, can only be evoked later on, in support of the diagnosis.

The course of a case of concussion varies. The functions of the brain may return to the normal within a few minutes, or at most within a few hours. If the circulatory disturbance has been very severe, death may ensue; or indications of gradually increasing brain-pressure or of brain contusion may supervene on the clinical picture of the concussion.

We have not yet discussed the condition which obtains between the sudden onset of the symptoms and their early decline, *i.e.*, the actual *symptoms of concussion*. There is hardly any single so-called "general symptom" which has been omitted from the descriptions of concussion. These include retardation and acceleration of pulse and respiration, headache, vomiting, loss of consciousness deepening to coma, excitement, pallor, &c. Order can best be introduced into this chaos by adopting Kocher's view that concussion is, in a limited sense, an expression of a sudden rise in cerebral pressure. The pressure curve in concussion would resemble a section from the curve of a slowly rising cerebral pressure, read backwards, descending from the severe to the slight symptoms. It depends, therefore, entirely on the stage in which we find the patient, whether there be signs of paralysis or of irritability, of pulse retardation or acceleration. The symptoms also vary from case to case, in accordance with the severity with which different parts of the brain are affected. If the medulla oblongata suffers most, the pulse and respiratory symptoms will predominate. Disturbance of consciousness will be most in evidence if the main stress has fallen on the cortex.

The following maxim will epitomize the diagnosis :—

Any cerebral symptoms manifested by a patient immediately after an injury to the skull—either of unconsciousness, of disturbance in the medulla, of irritability or paralysis—point to his suffering from "concussion of the brain." We cannot tell whether there be any further mischief, for which we must await subsequent developments. Such a statement will save both ourselves and the relatives from being consoled with a "simple concussion," while the patient shortly succumbs to pressure on the brain. The "further mischief" refers, as already stated, to brain contusion on the one hand, and meningeal hæmorrhage, with gradually increasing pressure, on the other hand.

(2) CONTUSION OF THE BRAIN.

We will now proceed to consider **contusion of the brain.** This consists of mechanical damage to the nerve tissue. This clearly differentiates it from concussion, which is a circulatory disturbance. But nevertheless there are numerous intermediate forms in which it is difficult to decide between the two, even at the autopsy, let alone during life. Experiment and histological research have shown that a severe blow causes, not only circulatory disturbance, but also mechanical damage to the nerve elements and interference with their mutual connections. Although no naked-eye changes be produced, the severest functional disorders may follow, and even death. The difference between these changes and foci of contusion visible at an autopsy is only one of degree. There is a whole series of connecting links between an obvious contusion and the microscopic changes which are present in what is clinically a simple concussion.

How can we clinically diagnose a contusion ? As in the case of concussion there is *immediate onset* of the symptoms after the injury. But the principal clinical difference between the two concerns the matter of *duration*. Again, in contusion focal symptoms predominate; there are signs of irritation or paralysis in cortical areas whose functions we know, whereas in pure concussion the "general symptoms" are more evident. But too much importance is generally attached to this distinction. There are many cortical areas whose functions we do not yet know, so that we cannot ascertain clinically whether they have been damaged. When a patient is unconscious, it is not possible to test whether all the areas are functional, as for instance the occipital cortex. Some of the slighter and of the moderately severe symptoms must really, as a rule, be referred to the accompanying concussion. A definite contusion of the medulla produces so rapid a death by paralysis of the vital centres, that no time is allowed for diagnostic reflections. But there is always one symptom which soon leads to an accurate diagnosis, and also permits the formation of a corresponding prognosis. This consists of a *persistent rise in temperature*, no matter whether we regard it as a sign of irritation of the corpus striatum or not. It is quite certain that a persistent and rising pyrexia coming on a day or two after the injury must be attributed to some form of brain contusion. Some fever is frequently present in cases of hæmatoma; but a temperature of 102° F. and above is very suspicious of contusion, even if a hæmatoma exists at the same time. This temperature does not presuppose an extensive contusion. Indeed the *post-mortem* appearances were almost negative in most of the cases wherein this fever was present; and we must, therefore, assume that microscopic damage of the brain tissue had occurred, death having followed too rapidly for the development of visible foci of degeneration. The diagnosis is easier if limited paralyses, and especially if limited spasms, remain after the symptoms of concussion have passed away. The diagnosis is equally easy if such symptoms come on *definitely*, while excitement and delirium continue, without presenting the features of brain pressure, to be subsequently described.

The following conclusion may be drawn for the diagnosis of contusion of the brain :—

A patient who, immediately after an injury to the skull, manifests brain symptoms, which persist for a whole day, and which do not fit in with the signs of a gradually increasing cerebral pressure, has sustained a contusion of the brain, in the widest sense of the term. This view is supported by the presence of irritative or localizing symptoms, arising from cortical areas of ascertained function. It is definitely confirmed by a high and persistent rise in temperature, which cannot be explained by infection from without.

We have hitherto maintained that the symptoms of contusion follow the injury forthwith. As a general rule this is quite true, but occasionally a simple contusion is accompanied by the typical symptoms of increasing cerebral pressure—a diagnosis confirmed by operation or autopsy. Obviously the contusion may lead to a persistent disturbance of the cerebral circulation, and thus to an increase of pressure. The brain behaves just like a contused limb—it swells up, and finds insufficient room within the skull, although there may have been no considerable effusion of blood. The following case is a good example of this acute traumatic swelling of the brain: A lad, aged 4, sustained a blow upon his head, and lost consciousness half an hour afterwards. Convulsions occurred on the right side, and deep coma came on immediately. There was a depressed fracture. Immediate operation showed that the cerebrospinal fluid was under high pressure, but no noteworthy hæmorrhage was found, although the brain was explored in three places. Cerebral pulsation had ceased, but this returned after a certain amount of cerebrospinal fluid had drained away. The cerebral symptoms disappeared and rapid recovery took place.

(3) CEREBRAL PRESSURE FOLLOWING INJURY.

The third form of damage which the brain suffers as a result of injury is the **rise in pressure consequent upon hæmorrhage.** The source of the hæmorrhage is a torn blood-vessel, either inside or outside the dura. As bleeding to the extent of nearly two ounces can be tolerated without any serious disturbance, it follows that the clinical signs of pressure will not appear at once, but only when the extravasation has reached a certain amount. *An interval* of varied length will intervene between this point of time and the moment of the accident. During this interval we may not recognize anything wrong with the patient, or may regard him merely as suffering from a rapidly recovering concussion. The duration of this "free interval" varies with the rate at which the blood pours out of the torn vessel. It may last from a quarter of an hour to several days—even a week or more. The symptoms consist partly of irritative, and especially of paralytic phenomena, dependent upon the part of the cortex pressed upon—*local cerebral pressure*—and also partly of manifestations of *general cerebral pressure.* The former obviously vary with the situation of the hæmatoma; but the latter usually run a regular course, in which the following stages can be distinguished :—

(*a*) Stage of *commencing cerebral pressure,* marked by signs of irritability, especially headache, psychical excitement and retardation of pulse.

(*b*) Stage of *completed rise of pressure,* marked by a combination of irritative and paralytic signs, and finally—

(*c*) The *paralytic stage* proper, when irritability has disappeared, and coma, Cheyne-Stokes breathing, and a rapid irregular pulse have supervened.

We may now briefly refer to the diagnostic value of the various so-called classical symptoms of cerebral pressure.

(1) *Headache.*—A continuous headache is the first symptom complained of, when the cerebral pressure is beginning to rise. There are many cases of injury to the skull wherein this is, and remains, the only sign of a slight hæmorrhage.

(2) *Vomiting,* as in all forms of cerebral pressure, is an important initial symptom. It is also present in injuries to the labyrinth, and it may be a transitory accompaniment of concussion. It is only of diagnostic value when, after a free interval, it heralds the onset of the other pressure symptoms.

(3) *The state of the consciousness* is of greater significance. The sensorium is not at first affected ; and if the hæmorrhage be slight, it may remain unaffected. But as the pressure increases, excitement and even delirium comes on ; when the pressure has risen high the signs of irritability abate, and the patient becomes sleepy, lapsing eventually into a persistent stupor, which in severe cases changes with terrible rapidity into complete coma.

(4) *Congestion of the Disc.*—Although this is the rule in pressure due to cerebral tumour, it is rarely met with when the pressure is due to hæmorrhage. It is an early symptom, which has often vanished by the time the patient is examined ophthalmoscopically. An error may arise in this connexion, if the injury has also damaged the blood-supply of the optic nerve.

(5) *The pupils* are rather contracted at first, but they react to light. One-sided dilatation, with absence of the light reflex, indicates severe damage—pronounced pressure—of the corresponding side. Dilatation of both pupils is a sign of the paralytic stage.

(6) *The Pressure Pulse.*—This term is used for the full, tense, and slow pulse of the early stage, although the paralytic pulse of the final stage is also a pressure pulse. The presence of this slowing of the pulse is of the greatest diagnostic value. But it may be absent, or at

any rate concealed, in cases of quite definite cerebral pressure. There are two possible reasons for this. Slowing of the pulse is, after all, a relative condition ; it must be estimated in relation with the other symptoms presented by the patient. For instance, if there is fever, a pulse of 70 or 80 will actually be slow, and represent a pressure pulse. The same consideration holds good, as Kocher remarks, when the breathing, for one reason or another, becomes rapid or deep. Finally, it must not be forgotten that a very high degree of local pressure may exist in an exposed skull fracture, without the development of any general brain pressure.

But, apart from this, the pulse may sometimes remain normal for quite a long time in cases of severe brain pressure, between the initial and the paralytic stages. Probably the presence of contusions may account for this.

Irregularity is not a necessary concomitant of the pressure pulse. It first appears when the patient approaches the paralytic stage. The pulse is first slow but regular, then slow and irregular, and finally rapid and irregular. This last stage is sometimes ushered in by a transition stage, wherein the pulse rapidly changes from slow to fast and *vice versa.*

(7) *Respiratory Changes.*—The breathing is at first hurried, but, like the pulse in definite cerebral pressure, it then becomes slow ; frequently it becomes deep, and with the further progress of the case irregular. In the paralytic stage, Cheyne-Stokes breathing appears.

(8) *The local pressure symptoms* begin with indications of irritability, viz., convulsions, contractures, increased reflexes, and later on paralysis of the centre exposed to the pressure. There will be mono- and hemispasms, mono- and hemiplegia, according to the situation and extent of the hæmatoma. Conjugate deviation of the eyes towards the healthy side in the stage of irritability, or towards the injured side in paralysis of the cortical-centre, is especially significant. ("In irritation of the centre the patient looks away from the lesion ; in paralysis he looks towards it.") If the hæmatoma is in the region of Broca's convolution, a more or less definite cortical motor aphasia will develop. If it is situated over the left temporal lobe, sensory, aphasia will be found : if in the occipital region (cuneus) there will be hemianopia, *i.e.*, blindness of the two halves of the visual field of opposite sides. Further details will be given in chapter VI.

All the symptoms will vary very considerably according to the situation and extent of the hæmatoma, and according to its rapid or slow formation. As the various centres differ in their irritability, it will often happen that, with the same pressure, one centre will be in a state of irritation, while another is already paralysed. It will, therefore, seldom happen that the symptoms described will be observed in their classical order. Nevertheless we shall not overlook brain pressure from injury, if we adopt the following summary of the foregoing :—

If a patient has sustained an injury to the skull, and then, after a free interval of very variable duration, or after the cessation of initial signs of concussion, brain symptoms of any kind supervene, it is probable that there is pressure from hæmorrhage, whether the brain symptoms be focal or general. If the symptoms increase we must regard the case as one of brain pressure, even if the classical signs of that condition are not present in a fully developed form, viz.: Disturbance of consciousness, slowing of pulse and respiration, and congested disc.

As previously remarked, there are cases of definite brain pressure after an injury, wherein there has been no considerable hæmorrhage at all. In the absence of any striking rise in temperature, pointing to contusion, it is sometimes quite impossible to be sure of the diagnosis.

If we are convinced of the existence of a hæmatoma, our next step is to determine its *situation*. As a rule we are led to a satisfactory conclusion by clinical experience, and by taking into consideration the position of the injury and the existing brain symptoms.

Experience shows that the hæmorrhages arising from the middle meningeal artery and situated between the dura and the skull, have a certain typical localization. Most frequently the bleeding indicates a rupture of the trunk of the artery or its anterior branch, and is situated beneath the squamous portion of the temporal bone. Less frequently, the posterior branch is torn, giving rise to a hæmatoma in the parietal region. More rarely still, a circumscribed hæmorrhage may be found over the frontal lobe, or in the posterior fossa.

A knowledge of the *site of the injury* is important for two reasons: because sometimes the hæmorrhage is immediately underneath, and because there may occasionally be an intradural effusion from contrecoup, at the opposite pole of the skull.

We have already seen how contrecoup fractures and corresponding brain contusions arise. Fig. 4 represents the same brain as figs. 2 and 3. Whereas contrecoup fractures are practically confined to the orbital roofs, contrecoup contusions with corresponding hæmorrhages may occur at any spot which constitutes the opposite pole of the force of the blow.

In the absence of other symptoms a careful search must be made for wounds or bruises on the skin which will indicate the site of the lesion and the probable situation of contrecoup effects. Large hæmatomata may be detected by percussion of the skull, because the note elicited is duller than on the healthy side.

The *existing brain symptoms* furnish the most important guides. Aphasia, monoplegia, hemiplegia, perhaps occasionally a hemianopia, indicate at once the site of the pressure. Localized convulsions or attacks of Jacksonian epilepsy will do the same. Often enough, it is necessary to employ all the three aids to establish an exact diagnosis.

For example: A young man was thrown off a horse, and on the following day he showed symptoms, partly of contusion and partly of brain pressure. As the symptoms increased, it was necessary to interfere. The skin was abraded in the right temporal region, and there was a bruise behind the right mastoid process. There was paresis of the right side of the face and the right arm, and the right thumb was spasmodically contracted into the hand. Conclusion: probably a hæmatoma over the site of injury, but certainly a hæmorrhage by contrecoup over the left hemisphere. Both sides were trephined and an insignificant hæmatoma was found on the right side under the injured skull, while on the left side a large intradural hæmatoma existed.

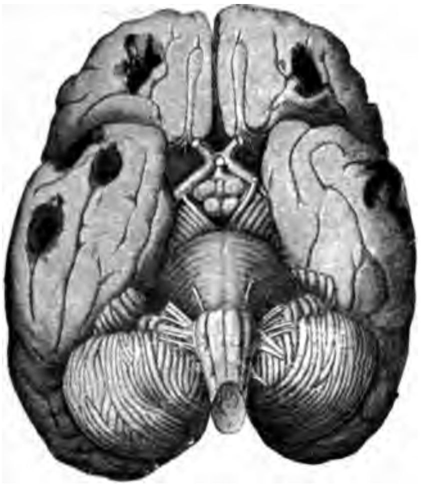


FIG. 4.—(After Kocher.) Contrecoup contusions from force applied on convexity; contrecoup fractures also produced. (See figs. 2 and 3.

A fixed dilated pupil is of very important diagnostic significance. We have invariably found the hæmatoma mainly responsible for the pressure symptoms, to be on the side of the dilated pupil—not on the opposite side, and we have often noticed the pupil contract, as the blood escaped and the cerebral pulsation returned.

Can we distinguish whether the hæmorrhage is *extra- or intradural?* This question has often been raised and has been variously answered. The following anatomical considerations will guide us: An extradural hæmorrhage arises from injury to the middle meningeal artery, a comparatively large vessel; while bleeding under the dura arises from injury of the much smaller vessels of the pia. Further, the dura is much more adherent to the skull than the soft membranes of the brain. We may, therefore, draw the following conclusion: Owing to the size of the vessel, an extradural hæmorrhage will develop more rapidly than the intradural, but owing to the close adhesion of the dura to the skull the former will remain more limited than the latter. For the same reason the focal symptoms of an extradural hæmorrhage will be more definite than those of the intradural variety. The latter is often characterized by a prolonged free interval, and by the predominance of general pressure symptoms over those indicating disturbance of the motor area. A hæmatoma situated directly over the cerebral cortex is more likely to produce irritation than one with an extradural situation. Intradural hæmatomata are more often accompanied by contusion, and therefore by rise of temperature, than are the extradural. Hæmatomata at the base of the skull are generally intradural, because the dura is so closely adherent to the bone in that region.

But the favourite situation for large intradural hæmatomata is on the convexity, in the neighbourhood of the falx cerebri. Even in this situation aphasia may be the only focal symptom which comes on eventually. I have seen this in two cases which came under observation shortly after one another. The free interval lasted four to six weeks, and there were no striking symptoms of interference with the limbs, despite a very extensive hæmatoma over the convexity. There were, however, slight aphasic disturbances, which, at any rate, indicated the side upon which the hæmatoma was.

Endeavours have been made to get information from *lumbar puncture*. Blood-stained fluid justifies the diagnosis of intradural hæmorrhage, but a negative result does not necessarily mean an extradural hæmorrhage. The decision as between an extra- and an intradural hæmorrhage is, however, not of much importance from the point of view of treatment. The main question to solve is whether there be a contusion or a hæmorrhage, and lumbar puncture affords no definite assistance in this direction. Blood-stained fluid may be expected both in contusion and in intradural hæmatoma. An *exploration* of the skull is much more valuable in a doubtful case. Sometimes a drill hole opening will suffice, but it is safer to make an opening—a few millimetres in extent—in order to ascertain with certainty the condition of the cerebral membranes and the cortex. Sometimes several such openings will be required before the hæmatoma is discovered. But this is a matter of therapeutics rather than of diagnosis, and should only be undertaken when all preparations have been made for an extensive operation. We do not wish, however, to imply that every case of brain pressure from hæmorrhage requires trephining. Many intracranial hæmorrhages — especially intradural—get absorbed without any interference, and we frequently see the symptoms of an incipient pressure disappear in a few days. But every patient with signs of pressure ought to be placed in circumstances wherein operation can be performed forthwith if symptoms increase, especially if any disturbance of consciousness occurs. But for this purpose it is necessary to recognize the pressure symptoms early, even without any exploratory trephining.

The sites of traumatic contusions and hæmorrhages are not always on the surface of the brain. Hæmorrhages have been observed in the medullary layer and in the ventricle. These deep-seated injuries are most liable to occur when the concentration of the force, transmitted through the brain, becomes divided, or when the force is diverted from its direct line, as happens in the vicinity of the ventricle, especially the 3rd, and the basal ganglia.

CHAPTER III.

ABSCESS OF BRAIN, HÆMORRHAGIC PACHYMENINGITIS, CYST OF BRAIN, TUMOUR OF BRAIN.

As one of the primary rules for the diagnosis of cerebral abscess or tumour, the proposition must be stated that occasionally both these diseases run a long course without a symptom, and then death may ensue with very few typical symptoms, and with such rapidity as to cause dismay, both to the relatives and to the medical attendant.

A young man, in perfect health, began to complain of pain at the back of the head. The doctor regarded it as an occipital neuralgia of unknown origin; treated the patient accordingly and, at his last visit, declared that the trouble was insignificant. Two days later, that is, ten days from the onset of the pain, the patient suddenly fell ill and died within a few minutes. The autopsy revealed a glio-sarcoma of the cerebellum, as large as a plum.

With cerebral tumours we must include growths from the meninges, the internal surface of the skull, as well as solitary tubercles and gummata, because all these diseases assert themselves by the same symptoms. These consist of a combination of symptoms due to general brain pressure with signs of local displacement and destruction. These symptoms also apply to cerebral abscesses, but the latter are distinguished from tumour-like formations by their cause, their rapid progress, and occasionally by high temperature.

The most important symptom, and the one which the patient notices first, is *headache*. In the early stages it only comes on in attacks, and there are long intervals of freedom. But its great intensity should serve to differentiate it from other ordinary headaches. Sometimes there are *attacks of giddiness*, or *epileptiform seizures*, in addition to the headache, before ever any permanent focal symptom can be detected by examination. Occasionally, the brain pressure reveals itself by apparently unprovoked *vomiting*, a symptom recognized as one of the early indications of tubercular meningitis. If the retina is examined, an indispensable proceding in all cases of severe, persistent, or intermittent headache, a *congested disc* will probably be already found at this stage. This, of course, absolutely excludes the possibility of simple neurasthenia. At first the disc is congested only on one side in tumours of the frontal lobe, and the congestion appears sooner in these tumours than in those of the motor area.

There is usually no *slowing of the pulse* in cases of abscess or tumour; it is only present when symptoms become acute.

It is necessary to utter here a word of warning which may be useful even to experts. Headache, attacks of unprovoked vomiting, and epileptiform seizures may occur during the course of *nephritis*, and an albuminuric retinitis may be confused with a congested disc. An examination of the urine should, therefore, never be neglected when there are brain symptoms of unknown origin!

Although a congested disc or optic neuritis *alone* must at once excite suspicion, it does not clench the diagnosis of cerebral disease, because it may also be present after infectious diseases, in syphilis and lead poisoning. In the latter case the optic neuritis may be associated with other brain symptoms of plumbism which would easily suggest a diagnosis of cerebral tumour if one were not aware of the cause, and the presence of the blue line on the gums. This possibility should always be considered. Error may also be caused by thrombosis of the central veins, which occurs in arteriosclerotic or anæmic persons.

A cerebral tumour of the central convolutions, or their proximity, gives rise usually to an epilepsy of the Jacksonian type, consisting of localized clonic spasms, which eventually merge into generalized attacks as the disease advances. But if the tumour is otherwise situated, the attacks may be indistinguishable from true epilepsy.

Having arrived at the diagnosis of a lesion which is causing pressure on the brain, we must distinguish between comparatively acute conditions, like *abscesses* and *hæmorrhages*, and those of more gradual progress like *gummata*, *tubercles*, *tumours* and *cysts*.

(1) ABSCESS OF THE BRAIN.

The principal argument in favour of abscess is the comparatively rapid course of all the symptoms. The events in tumour formation, which are spread over months or years, are often in the case of abscesses crowded into a few weeks. But there are many exceptions. Abscesses which are not very acute may remain unnoticed for months or even a whole year, if they have developed a firm limiting membrane.

Even in these chronic cases the whole course of the disease differs from that in tumour. The development of an abscess is more irregular than that of a tumour, as indicated by periods when the whole conlooked upon adiposis dolorosa as a pituitary symptom, and this view has again been put forward of late by Froelich and others. If this assumption were correct in the present case, it would follow that temporal hemianopia would be present in the right eye, which was not yet completely blind. As a matter of fact, it was found that this symptom could be clearly demonstrated, and in addition both optic

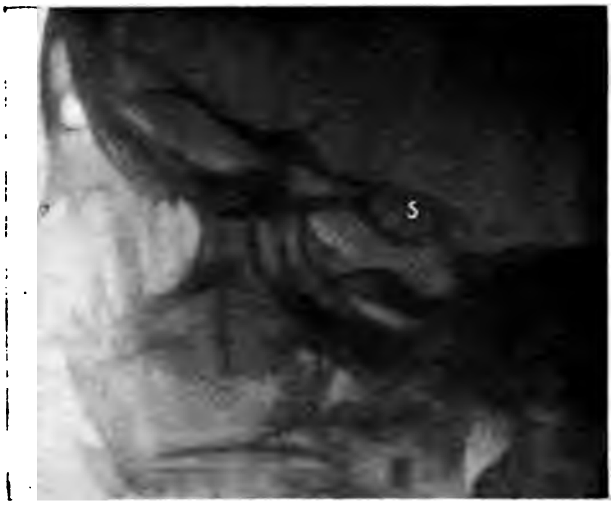


FIG. 5.—Normal sella turcica

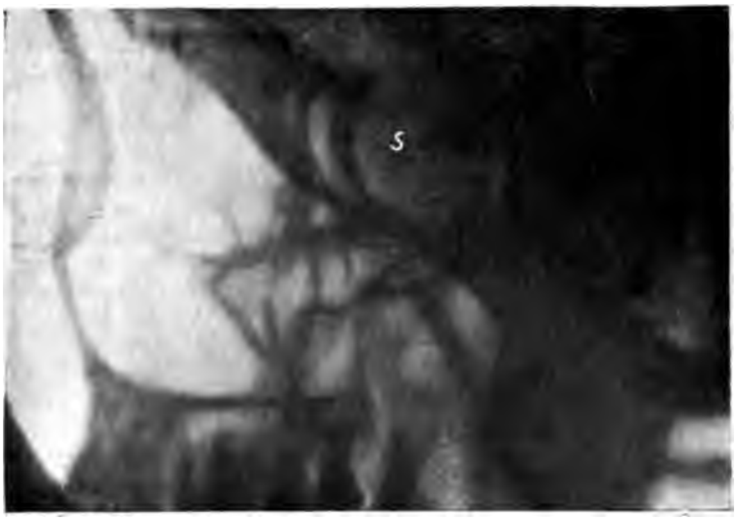


FIG. 6.—Widening of sella turcica in a case of pituitary tumour

nerves showed signs of atrophy. Thus the diagnosis of pituitary tumour was established. The slow growth of the tumour indicated that it was not a sarcoma, but probably a very slowly progressive adenoma, comparable to a goitre of the thyroid gland. A skiagram showed, in confirmation of the clinical signs, a great widening of the sella turcica.

Innocent tumours of the pituitary body, just like those of the thyroid gland, are characterized by the tendency to the development of cysts. This may explain the varying state of the visual disturbance which is occasionally noted in the clinical picture of pituitary tumour.

I have purposely refrained from mentioning puncture of the brain to determine the nature and situation of a tumour, as recommended by Neisser. The procedure is too dangerous and its results are too doubtful for it to be regarded as of diagnostic assistance to the practitioner. It may, however, be a useful expedient for the surgeon, when everything is ready for an operation.

Lumbar puncture is of great importance for the detection of tubercular (lymphocytes, tubercle bacilli) and gummatous (lymphocytes, Wassermann's reaction) processes. A negative result of the examination of the cerebrospinal fluid is not, however, conclusive. If the fluid has a yellowish tint and its albumen content is increased, suspicion of organic disease should be aroused. A rise of pressure over 20 cm., with the patient in a recumbent position, is of serious omen, and sometimes causes sudden death in tumours of the posterior fossa of the skull.

Röntgen-ray examination is harmless. It will reveal calcified or osseous tumours, and confirm the diagnosis of pituitary tumours by showing a widened sella turcica. (Figs. 5 and 6.)

CHAPTER IV.

THE CEREBRAL COMPLICATIONS OF SUPPURATIVE OTITIS MEDIA.

ALTHOUGH all the cerebral complications of otitis media do not originate from *mastoid abscess,* nevertheless it is their principal cause. It reminds us that the otitis is no longer a minor malady, but that it has reached a stage wherein danger to the brain is to be apprehended —a danger which can be prevented by timely treatment. It is the habit of aural surgeons to distinguish between acute and chronic mastoiditis, but as the *clinical* symptoms of both varieties are identical, we shall consider them together.

We must clearly understand that we are not clinically diagnosing

As the acute exacerbations often come on after a fall, the following medico-legal questions are important :—

(1) Did the patient fall because he had a hæmorrhage, or was the hæmorrhage the result of the fall ?

(2) Assuming that there was an accident, and a hæmorrhage occurred, was the latter due to the accident or to the antecedent alcoholism ?

After considering all the data of the previous history and the special circumstances of the "accident," an expert can often arrive at a definite opinion ; but nevertheless these cases are a fruitful source of litigation.

We have previously referred to inter-dural hæmatomata which come on late, and which improve when the effusion of blood is drained away. These cases must not be regarded as alcoholic pachymeningitis. The deposits on the internal surface of the dura are much slighter than in primary pachymeningitis.

(3) TRAUMATIC CYSTS OF THE BRAIN.

The diagnosis between tumour and *traumatic softening or cerebral cyst* is difficult. The symptoms may be so much alike that only the history supplies the decisive factor. If the patient is able to report a severe injury to the skull, evidence of which we can find on the scalp, the lines to proceed along are generally clear. It will be necessary, of course, to exclude a chronic cerebral abscess as far as the progress of the case and the temperature chart permit. But in the absence of any injury to the skull, the matter becomes more difficult. A most careful examination of the skin covering, and of the surface of the skull is indispensable. But as *tumours* may follow injury, one should realize a further fact before diagnosing a cyst, *i.e.*, the duration of symptoms—especially epilepsy—for years, without the supervention of new indications, and without any essential change in the patient for the worse.

A man, aged 54, was admitted to hospital for epileptic attacks. The symptoms, principally a right-sided hemianopia, pointed either to a cerebral tumour or a traumatic cyst of the brain. A careful examination of the skull, however, revealed a small scar over the left occipital region. It was not until then that the patient remembered a fall on the head off a hay cart in his youth. The diagnosis was, therefore, a traumatic cyst of the brain, which was confirmed by the operation.

Sometimes we may have to rely on an obstetric injury by forceps. If no history of any kind, pointing to an injury, is available, it is impossible to diagnose a cyst. But this is of no importance, so far as treatment goes, because a tumour which can be localized ought to be operated upon, if surgically feasible.

Traumatic cephalo-hydrocele, a special variety of cerebral cyst, is referred to later on.

Another special variety of cerebral cyst consists of non-traumatic, separate cystic accumulations in the soft meninges, especially in the vicinity of the cerebellum. Like the corresponding changes in the spinal cord, they possess all the symptoms of tumour, and their diagnosis can only be established at the operation. This condition is termed *meningitis serosa circumscripta.*

(4) NEW GROWTHS AND TUMOURS OF GRANULATION TISSUE.

If there is absolutely nothing pointing to abscess, pachymeningitis or cyst, we have to think of a **growth within the skull,** viz., *neoplasm tubercle* or *gumma.* As already stated, the possibility of a new growth is not excluded by a preceding injury.

This is well illustrated by a case described by Friedrich, wherein an injury gave rise to the development of a fibroma in the frontal lobe, the essential symptoms being of a psychical character.

If we could infer the nature of the suspected growth from the clinical signs, the diagnosis would be ideal. This is easy enough, in the presence of a malignant growth in some other situation, when the brain tumour is in all probability an example of metastasis. But in the absence of some such indication the task is much more difficult. The most important consideration for the diagnosis is offered by the general course of the illness. As a rule a *tumour* grows steadily on, whereas a *gumma* presents intermissions in its course, which, therefore, will probably be irregular and capricious.

The previous history and the systematic examination of the patient will afford us more information. Careful attention must be paid to everything which may give a definite lead to the diagnosis, without seizing exclusively on any one point. Severe, rapidly and steadily increasing symptoms will therefore suggest a malignant growth, even if the patient has a tubercular family history. The fact that the patient has had syphilis will not preclude a diagnosis of tumour or tubercle, if there is definite evidence pointing thereto. Again, a patient who has for some time been exhibiting symptoms of tumour, but has just recovered from syphilis, is obviously not the subject of a gumma.

Too much importance is not to be attached to a negative history of syphilis, in view of the fact that patients may be genuinely ignorant of having suffered, especially if they have been victims of "lues insontium." A positive Wassermann reaction is very significant, though it only means that the patient has had syphilis and is not completely cured, and the tumour may, after all, be a tubercle or a sarcoma.

The final, and often the decisive diagnostic point, is the effect of specific treatment. If energetic treatment, not unduly prolonged, is unsuccessful, or if its effect is doubtful, the patient must be regarded as a "surgical case," provided any focal symptoms are evident, no matter whether our diagnosis be tumour, tubercle or traumatic cyst. This leads us to the problem of *localization.*

Signs of paralysis are of most value, because these give a more correct indication of the seat of pressure than do symptoms of irritation, which may have their origin in the regions adjacent to the lesion. Our principal care must be to determine whether the tumour is in the cortex or involves the deep fibres or nuclei at the base of the brain. Thus hemiopia must not, at once, lead us to the conclusion that the growth is in the cuneus, for it is much more likely to originate in the optic radiation or optic tract. If it is possible to excite the pupillary reflex from the blind half of the visual field—the so-called hemianopic pupillary reaction—the tumour is probably in the visual area. If this reaction is not present it is an argument against that localization. To test this requires the full consciousness of the patient, and also a certain amount of intelligence, which are not always present in cases of cerebral tumour. We must, therefore, look for assistance from other symptoms, and basal symptoms will almost certainly be in evidence when a tumour is pressing upon the optic tract, as the following case illustrates :—

A young man, free from syphilitic or tubercular antecedents, fell ill with attacks of giddiness and headache. Later on, he also suffered from transitory mental disturbance and aphasic manifestations, for which he was brought to the hospital. A careful examination showed right-sided hemianopia, paralysis of the left oculomotor nerve, very variable speech disturbance, and intermittent mental derangement. It was not possible to obtain the hemianopic pupillary reflex with any certainty, but the discs were severely congested on both sides. The diagnosis appeared to be a tumour of the left frontal lobe, towards the base, pressing on the optic tract. This was the only explanation which would embrace all the chief symptoms—hemianopia, speech disturbance, and paralysis of the ocular muscles. The autopsy revealed a sarcoma as big as a fist occupying the left frontal lobe and growing towards the base. The left optic tract was compressed into a strip as thin as a piece of paper.

It is always necessary to seek for a diagnosis which will elucidate all the symptoms simultaneously. A localization which assumes several foci of disease is either wrong or if well attested must also assume a disease wherein the lesions may be multiple, like gumma, tubercle or secondary growths, and not merely a single new growth.

The details of cerebral localization are given in Chapter VI, but we append here a brief outline of the diagnostic symptoms of tumours, according to their localization. The same symptoms naturally hold good for abscesses and cysts. (*Compare* figs. 10-13).

Frontal Lobe.—Psychical disturbances, mental disease, sexual perversion, loss of memory, fine tremor of same side, hemiparesis of opposite side, ocular paralysis (extrinsic muscles), compression of optic tract, unilateral congestion of disc, aphasia frequent when tumour is on left side.

Region of Pre-central Sulcus.—Signs of motor irritability or of paralysis on opposite side. Extent of symptoms depends upon extent of tumour.

Parietal Lobe.—Disturbances of sensation. When of considerable extent symptoms arise from adjacent area, such as motor disturbance, signs of acute irritation, hemianopia, and, if the tumour be on the left side, sensory aphasia, word-blindness and agraphia.

Temporal Lobe.—Sensory aphasia (left side).

Occipital Lobe and Cuneus.—Symptoms of optic irritation, homonymous hemianopia, mind-blindness.

Cerebello-pontine Angle.—Trigeminal neuralgia, tinnitus, deafness, labyrinthine giddiness, paralysis of adjacent nerves, especially the sixth and seventh.

Region of Pons.—Spastic paralysis of the opposite limbs. Paralysis of sixth and seventh nerves of same side.

Cerebellum.—Pain back of neck, giddiness, early onset of double optic neuritis, rigidity of posterior cervical muscles, cerebellar ataxia (tendency to fall towards side of tumour), vertigo, nystagmus, forced movements, distant effect on contiguous nerves.

Pineal Gland.—Disturbances of the ocular muscles (owing to pressure on the region of the corpora quadrigemina). Early onset of puberty, both physically and mentally. Premature development of sexual organs, obesity, polyuria. Atrophy of genital organs in adults.

Pituitary Gland.—Pressure signs on optic chiasma, *i.e.*, bitemporal hemianopia, ending in complete blindness. This symptom is almost pathognomonic of a pituitary tumour, in the absence of such a cause as a fractured skull or a gunshot wound. There are often associated symptoms dependent upon the functions of growth, attributed to the pituitary body, *i.e.*, *acromegaly* on the one hand (the result of hypersecretion), and on the other a form of general *obesity* accompanied by defective genital development (typus adiposo genitalis), the result of deficient function. We are not clear as to the respective effects of disease of the anterior glandular part, and posterior nervous part of the pituitary body. An example of the latter variety will show how we arrive at the diagnosis of a pituitary tumour :—

A boy, aged 10, had been observed to squint since he was 2 years old, and from the age of 4 there had been defective vision, which culminated in complete blindness of the left eye. On examination, his appearance was very suggestive of myxœdema, especially his dry scaly skin and defective genital development. But, in contrast to what generally obtains in hypothyroidism, his intelligence was normal. His puffy appearance was more definitely due to deposits of fat than is the case in myxœdema. The thyroid gland was present, but was rather small. The possibility of the presence of pituitary disease was entertained, especially as his mother said that he was particularly sensitive to blows or touches all over his body. Dercum, years ago, a suppurative catarrh of the mucous lining of the mastoid antrum, but its resulting consequence. As long as the pus can freely escape from the antrum we have no means of diagnosing a mastoiditis. Comparative transillumination and percussion may guide us occasionally, but it cannot establish the diagnosis. It is only when the pus is retained, and damage occurs to the bone or periosteum, when caries develops or a sequestrum forms, that definite clinical signs appear which proclaim the course of the disease. This holds good whether the *otitis* be acute or chronic. Very many apparently trifling cases of otitis are accompanied by suppuration within the adjacent antrum, without our being aware of it. This suppuration is comparatively harmless as long as it does not lead to the results just mentioned. That the recognition of these sequelæ is important is obvious enough from Pitt's voluminous statistics, which show that the cerebral complications of otitis are responsible for ⅔ per cent. of all deaths.

As most of these cases result from chronic otitis, it is clear that our *first* diagnostic task is to recognize those cases of chronic otitis media which may be rescued by timely surgical treatment from subsequent complications of a severe character. If we only diagnose that our patient has a "chronic otorrhœa," our services will not have been of much value. Neither does a diagnosis of "polypus of the ear" get to the root of the matter. If we say that there is chronic suppurative catarrh behind the polypus, and that something is keeping up this catarrh, we shall be more nearly approaching the truth. If we know that the discharge is frequently fœtid, that the child often complains of headache, and if the region behind the concha is occasionally painful on pressure, we should not be content with diagnosing otorrhœa or polypus, but must inform the relatives that a serious disease is present—a disease with dangerous features, which, however, can be obviated or rendered harmless by operation. If the aural discharge has lasted many years, the term *cholesteatoma* is often applied to the malady. This is not really a tumour, but merely the result of a chronic desquamative inflammation.

It should be said that there are tumours which correspond to the "cholesteatoma" of the older pathologists, but these have little concern with the diseases of the ear.

The following is a typical case of the ordinary aural cholesteatoma:—

A little boy with an old-standing ear discharge was admitted to the hospital on account of an aural polypus. The discharge was offensive, the region behind the ear sensitive, but neither swollen nor inflamed. There were no acute symptoms, temperature was normal, and the general condition was good. Diagnosis: *Cholesteatoma.* The operation revealed a cholesteatoma larger than a pigeon's egg, bathed in fœtid liquid, which had exposed the dura mater to the extent of 1½ sq. cm.

Sometimes such a condition lasts a whole lifetime. I have seen a cholesteatoma excite brain symptoms forty years after the start of the aural suppuration.

There are two further points which support the diagnosis of cholesteatoma. The first is, that most cases follow the very destructive scarlatinal form of otitis media, and the second is, that the perforation of the drum is not central, but in the upper segment. Experience shows that cases of chronic otitis media with a central perforation are less likely to lead to cerebral complications and recover more easily, after appropriate treatment, than cholesteatomata, which always require surgical measures.

Our *second* diagnostic task, both in acute and chronic otitis media, is to recognize forthwith the onset of severe mastoid complications, in order to prevent, by means of a timely operation, the extension of the infection to the brain. When the infection is severe, especially after scarlet fever, a decision must be made within a few hours; days cannot be afforded for the purpose. It is easy to overlook the beginning of a mastoid inflammation in a child who is already very ill, but this oversight may cost its life.

A sudden *cessation of the discharge*, with a simultaneous *aggravation of the subjective symptoms*, despite the existence of a perforation of a drum, should arouse suspicion. Increase of the pain indicates retention, and the open perforation shows that the retention is deep within the adjacent antrum.

But persistence of the discharge by no means excludes some retention. The pus may escape from the tympanic cavity, and yet some may be dammed up in the accessory spaces.

Our view is confirmed if we obtain, on comparing the two sides, definite *tenderness of the mastoid processs on pressure*, provided that our examination is properly conducted. We may be deceived by a painful swelling of the glands situated over the mastoid, which may enlarge after a superficial inflammation of the external auditory canal. But careful palpation will avoid any difficulty on the score of these painful glands, because of their limited outline and because the bone beneath is not tender on pressure. If the inflammation of the external auditory passage is intense (diabetes) the periosteum in relation with it may become tender on pressure, without any suppuration in the air cells being necessarily present. Tenderness limited to the posterior portion of the mastoid process is not, as such, a conclusive proof of a mastoiditis. It is one of the chief symptoms of phlebitis of the emissary vein of the mastoid, and is only an indirect indication of suppuration within the mastoid cells; but, of course, it equally demands operation.

The remarks concerning tenderness on pressure apply also to the *swelling* of the soft tissues which cover the bone Inflammation of the lymphatic glands and phlebitis may each be a source of error, but if the possibility of these be borne in mind, no mistake is likely to arise.

A definite bulging of the ear away from the head (fig. 7) is an important sign of mastoiditis.

A *reddening of the skin* gives unmistakable support to the diagnosis, as long as it has not been caused by painting iodine or applying a blistering plaster.

Must all these signs be present to justify the diagnosis of mastoiditis? Those who have often operated for mastoid disease know that there may be severe suppuration, and even sequestrum formation, in cases which have presented but little tenderness, and which have had neither swelling nor reddening of the mastoid process. We must therefore take the other symptoms into consideration, principally the temperature and subjective feelings of the patient. If the original symptoms of an acute otitis have subsided, and then there is another rise of temperature accompanied by pain behind the ear, or if these symptoms appear during the course of a chronic otitis, we may infer the presence of mastoid disease, although the external visible signs may be indefinite. We may explain facial paralysis following aural suppuration in the same way. It should not be forgotten that, exceptionally, mastoid disease can occur without suppuration in the tympanic cavity, as in diabetes.

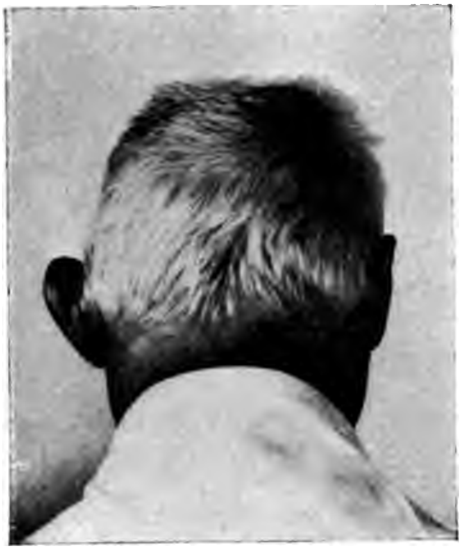


FIG. 7.—Bulging of the ear in mastoid inflammation.

The origin of the inflammation is also of diagnostic interest. If it has arisen in the course of scarlet fever, diphtheria, tonsillitis or measles, it usually means extension along the mucous membrane, and streptococci are the chief offenders. If the otitis is a sequela of pneumonia, the probability is that a hæmatogenous pneumococcic process has occurred. If the disease has come on insidiously and is obstinate in character, it is always significant of tuberculosis, more especially if facial paralysis supervenes without any acute symptoms.

If cerebral complications have supervened, our *third* diagnostic task is to determine their character accurately. Two practical questions are of urgent importance, viz., are the observed symptoms only sympathetic and reflex, or are they due to anatomical changes within the skull? If the latter, do these represent an epidural abscess, a cerebral abscess, meningitis, or thrombophlebitis of the transverse sinus?

The life of the patient depends upon the correct answers to these questions.

It is obvious that an acute suppurative process in close proximity to the brain has some effect thereon. Every enclosed focus of infection is surrounded by a zone of *circulatory disturbance*, and in the case of a focus in the ear the neighbouring area of brain and its sheaths may be involved. This explains the existence of a unilateral congested disc without any further changes within the skull.

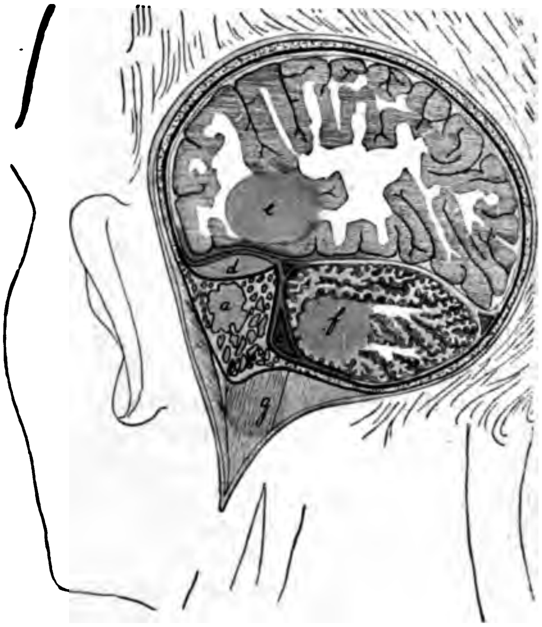


Fig. 8.—Diagram of inflammatory complications in suppuration of the temporal bone area. Green = pus; red = inflamed meninges; blue = venous sinus; violet = thrombosed sinus. *a* = mastoid antrum; *b* = mastoid process cells; *c* = thrombosed transverse sinus; *d* = subdural abscess; *e* = abscess in temporal lobe; *f* = cerebellar abscess; *g* = abscess under the sterno-mastoid (Bezold's abscess).

In addition to local circulatory disturbances, there are the pure *reflex processes*, which are especially well marked in children, and the symptoms of a *general intoxication*. Delirium, convulsions, and even rigidity of the neck, when they occur in the first acute onset of an otitis, or in mastoiditis, must not forthwith be put down to commencing meningitis. We should be content with opening the local focus of suppuration in the tympanum or in the mastoid process, and carefully watching the course of events. Actual cerebral complications take a definite time to develop, usually a few days. They *do not accompany* the first aural symptoms, not even in rapidly acute cases; but they *follow them*. But if such symptoms persist after the mastoid process has been properly drained, or if they occur afresh, then not only are we justified in diagnosing an intracranial complication, but we are compelled to do so and to act upon it.

We have hitherto been dealing with the matter from the point of view of an acute onset of otitis or mastoiditis. But this is not an invariable aspect. Discharge from the ear—chronic otitis—may have been present for years. Occasionally there is a temporary cessation of the discharge and the patient feels a dull pain deeply within the ear. The thermometer indicates a slight rise in temperature, but the patient, accustomed as he is to his malady, consults neither a thermometer nor a doctor. He knows that the discharge will soon start again, and he is content. But a fortnight or three weeks after this scarcely noticed incident, there supervene headache and giddiness, without anything remarkable occurring in the ear. Perhaps the patient may forget to tell the doctor that he has been suffering from an aural discharge. Such cases as these are really more serious than those which exhibit convulsions and delirium during an acute otitis.

What is happening within the skull? Are we dealing with an *epidural abscess*, a *meningitis*, a *cerebral abscess*, or a *sinus thrombosis?*

The condition of least gravity is that of **epidural abscess,** *i.e.*, a collection of pus between the roof of the petrous portion of the temporal bone and the dura mater, or more rarely posteriorly, between the petrous and the transverse sinus. These abscesses may be so free from symptoms that they are discovered quite accidentally when opening the antrum for mastoiditis. It is perhaps not correct to term their discovery "accidental," because the experienced surgeon will inspect the roof of the antrum in every case wherein he opens the mastoid process. If he has any suspicion of epidural abscess he will not hesitate to expose a limited area of dura, so as not to miss it. If the abscess is large, it will cause slight pressure symptoms: headache, somnolence, a somewhat slow pulse and a typical pus temperature. Really severe brain symptoms do not usually occur.

It is very exceptional to find any symptoms of local pressure, and then only in children. These concern the adjacent cortical areas and cause aphasia when the lesion is on the left side, and motor disturbances when the accumulation of pus is very extensive.

If the symptoms do not unanimously point to a simple mastoiditis, our first thought should be of an "epidural abscess," although the condition of the patient may not appear to us to be particularly disquieting. We may console ourselves and the relatives with this diagnosis, and yet we must confess that the pus may, after all, be on the other side of the dura. So that anyone who explores for an epidural abscess must be prepared to extend his search, if necessary, beyond the dura into the brain substance.

But are there no symptoms which enable us to diagnose a cerebral abscess forthwith ? Let us first consider the fairly common **abscess of the temporal lobe,** which damages the same cortical area as an epidural abscess, when in its usual position on the roof of the petrous bone. Theoretically, we may anticipate, from a temporal lobe abscess on the *right* side, a diminution of auditory perception in the left ear. But as the right ear is not available for comparison, apart from the fact that both ears are often affected in otitis, we must discard this test. An abscess in the *left* temporal lobe may cause sensory aphasia, but does not always do so. Although it is conceivable, as already stated, that an extensive epidural abscess may cause a certain measure of aphasia, owing to pressure upon the temporal lobe, nevertheless a definite sensory aphasia must be put down to the credit of a cerebral abscess. But in the absence of aphasia, or when the disease is on the right side, we must draw our conclusion from the intensity of the symptoms. If the headache, slow pulse and vomiting are very marked, or if unconsciousness be present, there is much greater probability of cerebral abscess than of a simple epidural abscess. The temperature can be relied upon as a guide. On the whole, an epidural abscess is more likely to exhibit the regular curve of a pus temperature than a cerebral abscess, wherein the temperature is quite irregular. Days with normal temperature alternate with sudden and steep ascents in the chart. One must not neglect the "impression" conveyed by the patient. A patient with an epidural abscess does not convey the impression of a severe case. The general condition of a case of cerebral abscess does not give rise to great anxiety, except in the last stage. Nevertheless a careful observer will detect "something" in the psychical state which, however, baffles definition. If the patient is a child the parents are quite sure that there is some "change," and a mother's observation is often more acute than that of a medical attendant. In diffuse *purulent meningitis* the clinical picture is quite different. The existence of a severe illness is in striking evidence. As this impression gains in intensity from hour to hour, the diagnosis no longer remains in doubt, for there is a steady progress in meningitis, whereas in cerebral abscess the course is fluctuating.

The following is a typical case of an abscess in the temporal lobe, following otitis :—

A little girl had been suffering from a discharge from the left ear for several years. She began to complain of severe pain behind the left ear three weeks before admission to the hospital. The mother thought that there was a slight swelling, but the pain ceased on the application of domestic remedies, and the child returned to school, apparently in normal health. Then, severe headache and vomiting set in, and the doctor noted, at that time, a slight facial paralysis of the left side. There was a little tenderness behind the ear, but no swelling, though an offensive discharge issued from the meatus. There was neither aphasia nor congestion of the discs; the pulse was not retarded, and there was only a little fever. The facial paralysis indicated a severe inflammatory process within the temporal bone, and the long duration of the disease, together with the offensive discharge, made the diagnosis of cholesteatoma very probable. The headache and vomiting made one think of abscess, but these symptoms may have been remote ones. The operation, which was undertaken immediately, revealed a cholesteatoma bathed in fœtid pus. The facial nerve canal, seen on the floor of the large bony space, was eaten away into an open channel. There was improvement for two days after the operation; then the headache recurred, with striking changes in the psychical condition and signs of aphasia. Congestion of the left disc was beginning, and therefore a diagnosis of abscess in the left temporal lobe was made. A second operation revealed an enormous abscess in this position, filled with fœtid fermenting pus. The aphasia disappeared, the mental condition became normal, and in a few weeks the child left the hospital, completely cured except for a remaining facial paralysis.

The diagnosis of *cerebellar abscess* presents greater difficulties. Just as a temporal lobe abscess is the consequence of the spread of the inflammation *upwards*, so is cerebellar abscess the consequence of its spread *backwards*. The bone is not necessarily destroyed, but the transverse sinus, which runs in the direction of the cerebellum, is frequently involved. If, therefore, signs of a cerebral abscess follow those of a sinus thrombosis, our first thought will be of the cerebellum. The only other symptom which points to this diagnosis is giddiness, of the character of cerebellar ataxia. But it must not be forgotten that any intracranial disease can cause giddiness, and that if the giddiness be very severe it may depend upon the labyrinth being affected. To make the diagnosis of cerebellar abscess probable, the cardinal signs of a cerebral abscess must be present, viz., headache, vomiting, and possibly slow pulse and congested disc, and in addition a giddiness which causes the patient to stagger, or even entirely prevents him from walking. If a sinus thrombosis is also present it supports the diagnosis, because it shows that the inflammatory process has extended backwards, but on the other hand this thrombosis may itself cause symptoms similar to those of a cerebellar abscess. It is therefore prudent to give the first signs of a thrombosis the opportunity of disappearing before proceeding in search of a cerebellar abscess.

The following is a classical case of cerebellar abscess:—

A young man, the subject of an old chronic otitis, suffered from an abscess behind the left ear. His medical attendant opened it by means of Wilde's incision, and rapid healing followed; but the patient began to complain of headache and giddiness a few weeks later. The pulse became slow and irregular, and definite signs of an abscess in the brain appeared. Giddiness was the only symptom of localizing value, and it was so intense that the patient, whose nervous system was otherwise healthy, could hardly walk. Operation, and unfortunately the subsequent autopsy, confirmed the presence of the expected cerebellar abscess.

How can we recognize **sinus thrombosis?** Its brain symptoms are of least significance. Of course, the blocking up and the infective inflammation of a large venous channel will produce a disturbance in the cerebral circulation which will manifest itself by certain signs, but these are so indefinite that no diagnosis can be based on them. There will be headache, vomiting and giddiness in cases of meningitis, just as there are in cases of abscess. But there are some special features which permit us to recognize disease of the sinus. These consist of changes in the veins connecting with the sinus. Frequently there is tenderness over the region of the emissary vein in the posterior portion of the mastoid process ; occasionally there may be œdematous swelling due to the spread of the thrombus externally, through the emissary vein.

Special attention must be paid to the condition of the internal jugular vein, which usually shares in the thrombophlebitis, at any rate in its upper section, which presents in its course some œdema, in the form of a cylindrical painful swelling. If these signs follow a previous aural discharge they suffice for a correct diagnosis. But this swelling behind the mastoid process must not be confused with inflamed glands or deep cervical abscesses, which are occasionally noticed after acute and chronic middle-ear suppuration, and which may spread thence over the entire side of the neck. I have seen an abscess of this kind—a gas phlegmon—reach down to the gluteal region. This variety of abscess, first described by Bezolt, may follow a purulent sinus thrombosis, but this is not its only cause.

If we suspect a sinus thrombosis in the absence of definite local signs, the diagnosis can be established in severe cases by the general symptoms of an infective thrombophlebitis. Repeated rigors followed by characteristic outbursts of perspiration, sudden elevations of the temperature up to 104°—106° F., a pulse rate of 140 or more, and the subsequent onset of pulmonary embolism or joint complications—all these form a clinical picture which once seen is never forgotten, and which even the inexperienced cannot fail to recognize.

The onset of infective disease within the sinus is not, however, invariably accompanied by such severe symptoms. Whether it follows infective thrombosis of small endosteal veins (Körner), or circumscribed peripheral thrombosis of the transverse sinus, it very frequently leads to a chronic form of sepsis, manifesting itself merely by inflammatory, evanescent exudations in connective tissue, muscles and joints. These exudations may become completely absorbed. The following is a typical case.

The patient, a female, aged 40 years, was referred for treatment because of osteo-myelitis in the left tibia. The soft tissues over the bone were infiltrated; but, on examination, the bone itself did not appear to be involved. Similar signs appeared temporarily on the other leg. There were irregular sudden rises of temperature, with some indications of rigors. A temporary otitis media had occurred four weeks previously. Clinical diagnosis: endocarditis, septicæmia probably due to streptococci, with evanescent non-purulent metastases. The blood revealed a number of colonies of a small hæmolytic streptococcus in every cubic centimetre.

The only thing which now remains is **meningitis.** If the symptoms do not fit in with any of the previously described conditions, we may diagnose it by a process of exclusion. The disease is ushered in by headache, stiffness of the neck, vomiting and fever. From the very beginning the aspect is grave, and the gravity increases from hour to hour. Sleeplessness alternates with delirium, and as the disease advances unconsciousness becomes more marked. Motor symptoms of irritation and paralysis may come on without any definite order, and in the most variable combinations, but, on the other hand, they may remain entirely absent. The fever persists, the initial slow pulse becomes rapid, the respiration interrupted, and death ensues in profound coma.

If the diagnosis is doubtful, it may be cleared up by lumbar puncture. A negative result would justify us in scarching for a cerebral abscess, but if the fluid is cloudy, operative interference is useless. The few cases of otitic meningitis which have recovered after trephining were of a circumscribed character. We must endeavour to operate before the infection has reached as low down as the lumbar spine, if indeed such infections are susceptible of recovery.

The above remarks on otitic meningitis, apply, *mutatis mutandis,* to all forms of acute purulent meningitis, especially to meningitis of the frontal sinus after cranial injuries.

CHAPTER V.

THE PROBLEM OF EPILEPSY.

THE surgeon occasionally sees all three varieties of epileptics. He is most frequently concerned with those who suffer in consequence of previous *injury*, then with those whose epilepsy depends upon a non-traumatic anatomical lesion of the brain, and finally **genuine** *epileptics* find their way to him when medical treatment can offer no relief. As the surgeon has to pronounce the final verdict in regard to operation in any given case, he must be able to decide to which class the patient belongs.

The first thing to decide is, whether the patient is really epileptic, and here the difficulty begins. We will assume that no gross diagnostic blunder has been committed, and that neither uræmia nor eclampsia has been mistaken for epilepsy. It is important, however, to recognize accurately the so-called "epileptiform" attacks, and those of "petit-mal," and to distinguish the violent epileptic seizures in hysterical conditions. The differential diagnosis of this last group of cases, which we cannot here discuss, is not always very easy, especially when the tongue is not bitten, when other self-inflicted injuries are absent, and when the moral perversity may be attributed to either one or the other.

As just stated, the surgeon's most frequent opportunities for seeing epileptiform attacks are in post-traumatic cases. The following is an instance:—

The patient was a young man of a low psychopathic type, afraid of work and prone to lying. He sustained a stab in the side with a knife; the wound healed rapidly, but he developed epileptiform attacks, which kept many observers in doubt for weeks as to their epileptic or hysterical character. He was sent from one hospital to another; he ran away at times and simulated, consciously or unconsciously, the most varied diseases. He wanted to be trephined, to have his thorax explored, to have a laparotomy performed, and he also indulged in attacks of mild self-injury, &c.

It really matters little whether such an afflicted individual is diagnosed as an epileptic or a hysteric or as a combination of the two. His motor functions, like the rest of his nervous system, are in a state of intense irritability, and a prudent surgeon will leave him severely alone.

But, assuming that the case is one of real epilepsy, we have to consider the *clinical history*, the *course of the attack*, and the *objective condition* during the intervals, before we can classify the patient correctly.

The *clinical history* must be investigated for all those circumstances which can point to the non-surgical character of the epilepsy, viz., heredity, alcohol, absinthe, lead intoxications, and infections, especially syphilis. But if an alcoholic suffers from epilepsy, it must not be assumed that *only* the alcohol can be credited with it, for it may be due to some accidental injury to the skull. Hereditary predisposition, intoxications, and infections form the basis on which epilepsy develops, when some exciting factor comes into play. This factor may be so trifling that it baffles observation—then the case is and remains medical. But this factor may be provided by the appreciable results of an injury, and then the case is surgical, in so far as these results can be removed by operation.

If the question of congenital or inherited predisposition is disposed of, we must first ascertain whether the patient has ever sustained an injury to the head, more especially an injury involving both the skull and the brain. Our inquiries must be prosecuted even unto the patient's birth, because epilepsy may be the consequence of a porencephalitis due to injury by forceps.

But, apart from forceps, childhood offers abundant opportunities for cranial injuries. If a child is not dropped by its nurse, sooner or later it will fall on its own account. The skull may be compressed without the attention of the parents being attracted thereto, and the brain may suffer severe damage beneath an apparently uninjured skull cap. This damage may, in its turn, produce softening, with consequent cyst formation or porencephalitis.

If it is clear that the patient has sustained an injury to the skull, we must ascertain whether the epilepsy is a direct or indirect result. This is especially important from the point of view of accident insurance. A patient may have been epileptic before his accident, but may wish to fasten the responsibility for his disease on the period following the injury. But even if it transpires that there has been a long interval between the injury and the first attack of epilepsy—years may have elapsed—we cannot conclude that the epilepsy is not traumatic. To cite one possibility, the injury may have caused a cerebral cyst, and this, many years subsequently, may declare itself by means of epilepsy and other symptoms. But as a rule traumatic epilepsy appears within a few months of the accident.

I once had a patient with a traumatic cyst in the frontal lobe. The lad began to masturbate eight years after the injury, and then epilepsy manifested itself in the form of "petit-mal" at first. It was quite clear that the individual attacks were a consequence of this habit.

So far we have confined ourselves to injuries of the head, or, rather, of the brain. But there is a form of epilepsy which may follow any peripheral irritation, apart from injury to the brain. This is called *reflex epilepsy.* Any peripheral injury may set up this external irritation and painful scars over bones, or those involving nerve-trunks, have long been credited with this evil. Even foreign bodies in the nose or ear may cause epilepsy. Demme reports a case wherein a rectal polypus acted in this way.

When I was a student I saw a case of Kocher's, in which typical Jacksonian epilepsy appeared to start from a scar in the hand. The excision of the scar proved useless. It was then decided to trephine, but he escaped from this project by dying suddenly. The autopsy revealed a well-circumscribed tumour in the situation selected for the operation.

Although too much has perhaps been attributed to reflex epilepsy, we must not neglect its consideration when examining the patient. Our conjecture will become a certainty if the patient experiences any abnormal sensations in the suspected scar before the attack, or if we are able to directly excite an attack by pressing on the scar.

A boy, aged 3, who had never been epileptic, was brought into hospital with definite attacks, a few days after a fall on his forehead. Examination showed an abrasion of the forehead, from which erysipelas was spreading, and a subjacent abscess. This was opened; there was no fracture of the skull. The fits ceased immediately, and the case, therefore, was either a toxic or reflex epilepsy.

The actual **observation of the fit** is an important part of the clinical history. If it resembles an ordinary attack, beginning with general convulsions, and not regularly affecting any particular region of the body, either by a preceding aura or subsequent effects, we can draw no conclusion regarding its etiology. General convulsions usually signify so-called true epilepsy, *i.e.*, epilepsy with an unknown anatomical basis, but they also occur in other forms of epilepsy, which are caused by certain gross anatomical changes.

It is quite different when the aura, or the post-epileptic paralytic phenomena, either persistent or transitory, indicate a definite area of the cortex as the seat of origin of the attacks. Two varieties may be distinguished in this connection. In one, the convulsions are limited to a circumscribed motor area, and we are therefore confronted with typical Jacksonian cortical epilepsy. In the other, the attack similarly begins in one definite area, but it marches along, in anatomical procession, into other areas, and thus the fit becomes generalized. Temporary paralysis in the area corresponding to the seat of origin may follow the attack.

The relatives can often furnish useful information on these points, but it is always advisable to personally confirm their statements, or to obtain their confirmation from a hospital-trained attendant, because our treatment must frequently be dictated by this information.

Finally, a careful investigation of the **condition of the patient** unconscious patient, the previously noted signs must guide us. They may be summarized thus: *A central lesion is indicated* (1) *when the eyes and the frontal branches are comparatively unaffected;* (2) *when there is an opposite paralysis after an injury; or* (3) *when the limbs are paralysed on the same side. A peripheral lesion is indicated* (1) *when all the branches are affected on the same side as the injury to the skull;* (2) *when ocular*

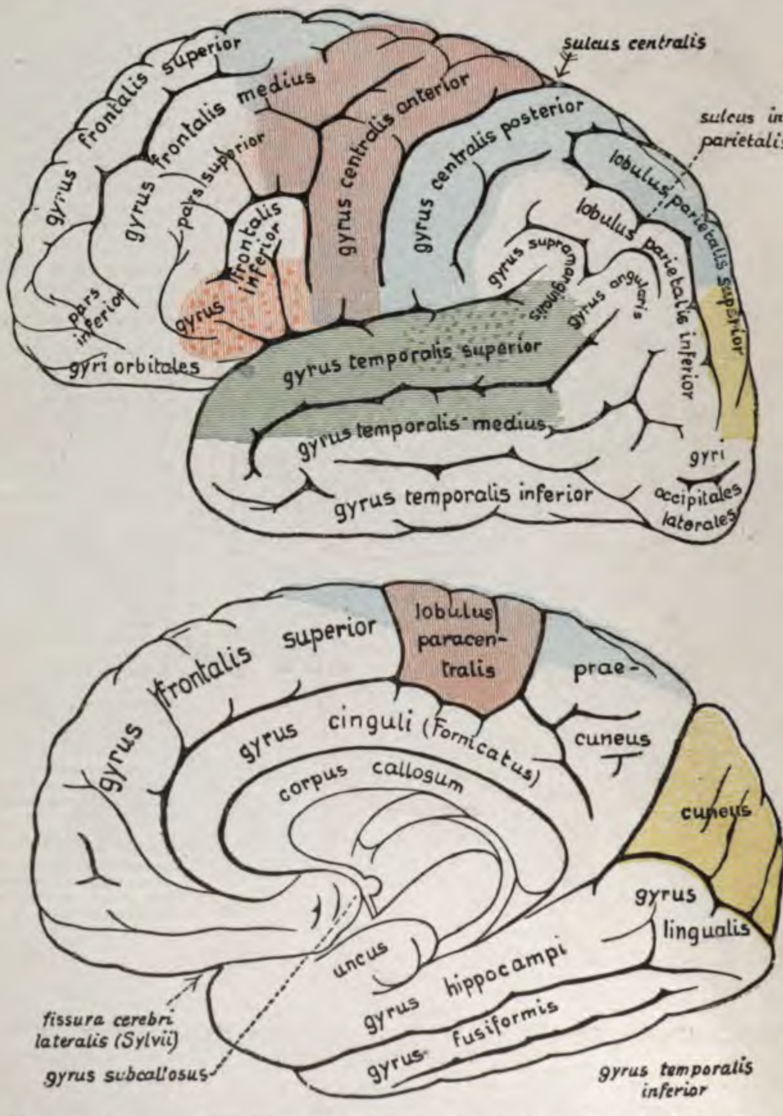


FIG. 12.—The most important cortical areas, as at present established. Red = motor area. Dotted red = motor speech centre. Blue = sensory area. Green = auditory area. Dotted green = sensory speech centre. Yellow = visual area.

anticipate benefit from operation. Unfortunately, experience shows that the hopes based on operative measures have fallen far short of anticipation. This is, however, not the place to discuss this circumstance. Nevertheless, interference is advisable in all cases of traumatic epilepsy, unless the long duration of the illness, or the great frequency of the fits, or the mental changes, indicate that the whole central nervous system is too deeply compromised. The prognosis in tubercle and in tumours depends upon the possibility of complete removal of the growth. Post-apoplectic epilepsy we must leave to the physician, and this also marks the limit of the assistance we can give in genuine epilepsy.

CHAPTER VI.

SOME REMARKS ON CEREBRAL LOCALIZATION AND FOCAL DIAGNOSIS.

We have already touched upon the subject of focal diagnosis—*i.e.* the detection of the situation of a brain lesion, based upon our modern knowledge of localization—but it is necessary to deal with it now in a more connected method.

Our first task is to distinguish a peripheral from a central lesion of the nervous system ; but, as we have seen in the case of a fractured skull or of a cerebral tumour, this is not always easy, because the two lesions may be present at the same time.

Let us begin with (1) **disturbance of vision.**

A glance at the very simplified diagram in Fig. 10 suggests the various possibilities which possess surgical interest. *Blindness of one side* indicates an interruption in the path between the retina and the chiasma (*f*), which may be caused by primary or secondary tumours of the orbit, or a fractured base. *Bitemporal hemianopia* is the classical sign of a tumour in the region of the chiasma (*d*), *e.g.*, a pituitary tumour. *Blindness of both sides* depends upon the same cause (*e*), but it indicates a later stage or a more extensive lesion (*e.g.*, gunshot with suicidal intent). It may also occur when there is severe congestion of the disc in cases of chronic cerebral pressure (tumours and cysts in any position, but more especially in the posterior cranial fossa). Finally, it may occur through a simultaneous lesion of both cortical visual areas—but this is rare (tumour of the falx cerebri). *Homonymous hemianopia* indicates a lesion in the optic tract (*c*), in the optic radiation (*b*), in the primary visual centres of the optic thalamus, lateral corpus geniculatum and anterior corpus quadrigeminum (*b*¹), or the cerebral cortex, (*a*), *e.g.*, tumours, cysts, or trauma.

From the surgical standpoint it is most important to distinguish between a lesion in the optic tract and in the cortex (see under Cerebral Tumour). A sign first described by Dufour may be of assistance. He states that if the visual path be interrupted at *c* or *b*, the vision is dimmed; if the lesion be in the cortex the vision is totally absent. The hemiopic pupillary reaction must be tested to determine the mobility of the pupil. If no reaction is obtained, the lesion is at *c*, thus excluding lesions both at *b* and *a*.

The cortical lesion is sometimes incomplete (*e.g.*, in tumours). In such cases the hemianopia is also incomplete, there being merely a homonymous loss of one-quarter of the visual field, which, however, must not be confused with a unilateral scotoma of peripheral origin. There still remains another form of visual disturbance, which requires brief consideration, although it rarely possesses surgical interest, as it is bilateral—*i.e.*, **mind blindness.** Whereas the destruction of the cortical area marked (*a*) in the diagram prevents the perception of visual impressions, these impressions still arise if the cortex of the cuneus is unaffected, but they will not be appreciated by the mind, if the fields for visual memory on both sides, or the association fibres leading to them, are destroyed. This latter condition is termed "mind blindness," in contrast to "cortical blindness," due to destruction of the cortex of the cuneus on both sides.

All these examinations demand normal intelligence and retained consciousness, but these patients frequently possess neither the one nor the other. We must then be content with the ability to distinguish unilateral blindness, bitemporal and homonymous hemianopia. The examination can be carried out with approximate accuracy without a perimeter, although a correct visual chart is always desirable.

We may infer from the pupil reflexes that they are served by their own special fibres. Their behaviour may be ascertained from the facts illustrated in fig. 10. Disturbances in the reflexes without simultaneous visual disturbances always suggest an isolated lesion of the oculomotor nerve or its nucleus.

(2) **Derangements of the extrinsic ocular muscles** can only be detected by causing the patient to look in definite directions, otherwise they may be confused with **conjugate deviation.** As soon as the patient is told to move his eyes, it will at once be seen whether he squints or has conjugate deviation. If there is no movement of the eyes when directed to look sideways, there is probably conjugate deviation. Whereas advanced ocular paralysis can easily be detected,

its slighter forms can be concealed by patients bringing the non-affected muscles into action. A very careful examination for double vision in all points of the visual field is therefore necessary. It has already been said, and it will subsequently be repeated, that ocular paralyses, except when they are of purely medical significance, indicate trauma, inflammatory diseases, tumours of the orbit or base of the skull, or affections of the base of the brain. Conjugate deviation, on the other hand, is always a sign of disease above the nucleus (the patient looks towards the healthy side when there is irritation, and towards the lesion when there is paralysis). In this connection, tumours, cysts, abscesses in the cortex, or in the sub-cortical white substance, and also injuries, are especially of surgical interest.

The nucleus of the sixth nerve forms an exception to what has just been said. According to the most recent researches, this nucleus represents a more deeply situated co-ordination centre. Injury thereof, which hardly possesses any surgical significance, leads to conjugate deviation towards the healthy (opposite) side.

In addition to conjugate deviation, *Nystagmus* must also be mentioned as a symptom of irritation. This may be excited artificially through the vestibular nerve, by injecting cold or hot water into the ear. This symptom is frequently associated with damage to the nerve itself, and also with tumours in the neighbourhood of the cerebellum. It is obviously also present in diseases in the vicinity of the nuclei of the ocular muscles.

(3) It is most important to understand that in a peripheral injury of the **facial nerve** (see fig. 11) all the branches to the face are equally involved; but when the lesion is central the lower facial branches suffer most, because the branch to the forehead is innervated from both sides of the brain. It must also be remembered that peripheral paralysis is on the same side as the injury, whereas a central lesion produces paralysis on the opposite side. This distinction is valuable when we know the situation of the injury to the skull, but discrimination must be exercised, because in cases of injury to the cortex by contrecoup the paralysis is on the same side as the injury. Signs of irritation, convulsions in the paralysed region, as well as paralysis of the limbs on the same side, denote a central lesion. Definite conclusions may also be drawn from the varying conditions of voluntary movement, associated movements, and those of facial expression. If these last are retained, and at the same time associated, and voluntary movements are completely lost, it is quite certain that the lesion is central. But we can infer nothing from the predominance of the voluntary over the associated movements, because this peculiarity may be present in a central as well as in a peripheral paralysis (Sahli). Paralysis of the *secretory and taste fibres* of the facial nerve, shown by a decrease of saliva and by diminution on the

corresponding half of the tongue of taste appreciated by the fifth nerve, is only of limited diagnostic value. Motor paralysis due to a lesion between the nucleus and the geniculate ganglion, really at the base of the brain (fig. 11*c*), is accompanied by taste disturbance only; but lesions in the petrous bone (fig. 11*d*, and *e*), below the geniculate ganglion, cause taste disturbance and also a diminution of salivary secretion; disturbance of lachrymation is only caused by damage above the geniculate ganglion.

a. Cortical lesion (cuneus, trauma, tumour, cyst, abscess). Homonymous crossed hemianopia (loss of opposite visual fields). Absence of vision complete. Reflexes normal.

b. Lesion of optic radiation, or of primary visual centres(*b*¹). Causes as above. Symptoms as in *a*, but vision dimmed.

c. Lesion of optic tract (generally tumour). Same symptoms as *b*, and *b*¹, but pupil reflexes from opposite visual fields lost (hemiopic pupillary reaction).

d. Partial lesion of chiasma (pituitary tumour). Loss of both temporal visual fields (bitemporal hemianopia). Corresponding loss of pupillary reflexes.

e. Complete lesion of chiasma (tumour, traumatism, especially suicidal gunshot wounds). Blindness of both sides, and complete loss of pupillary reflexes.

f. Lesion of optic nerve (trauma, tumour). Unilateral blindness, with loss of reflex on the diseased side, and of consensual contraction on healthy side. If lesion is incomplete, unilateral scotoma, or concentric contraction of visual field.

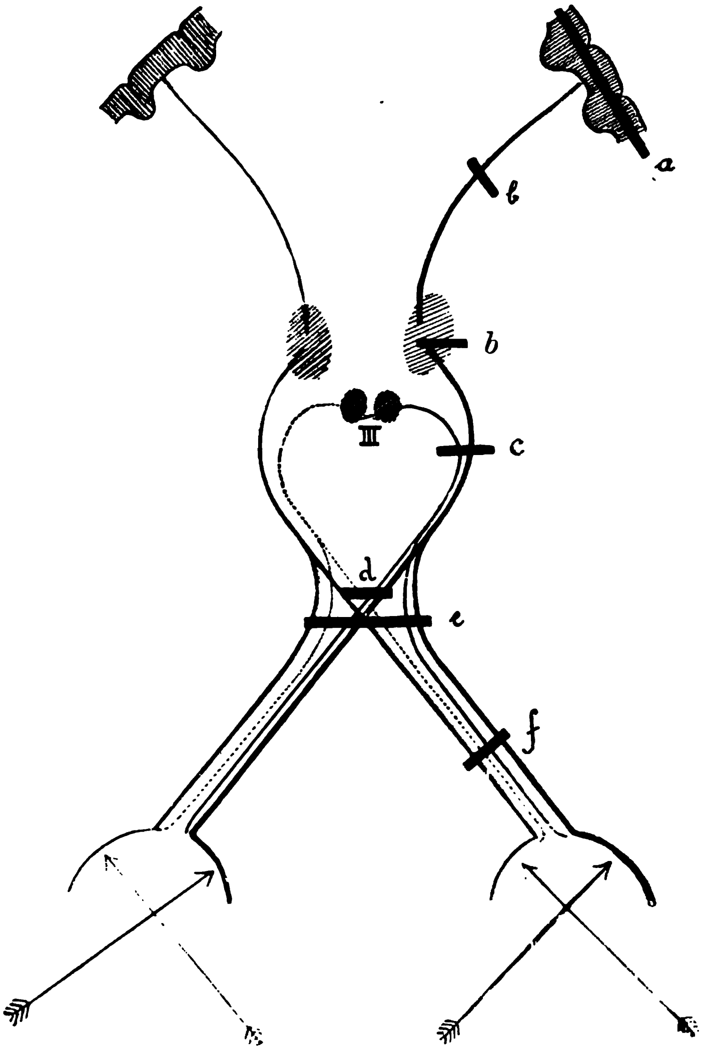
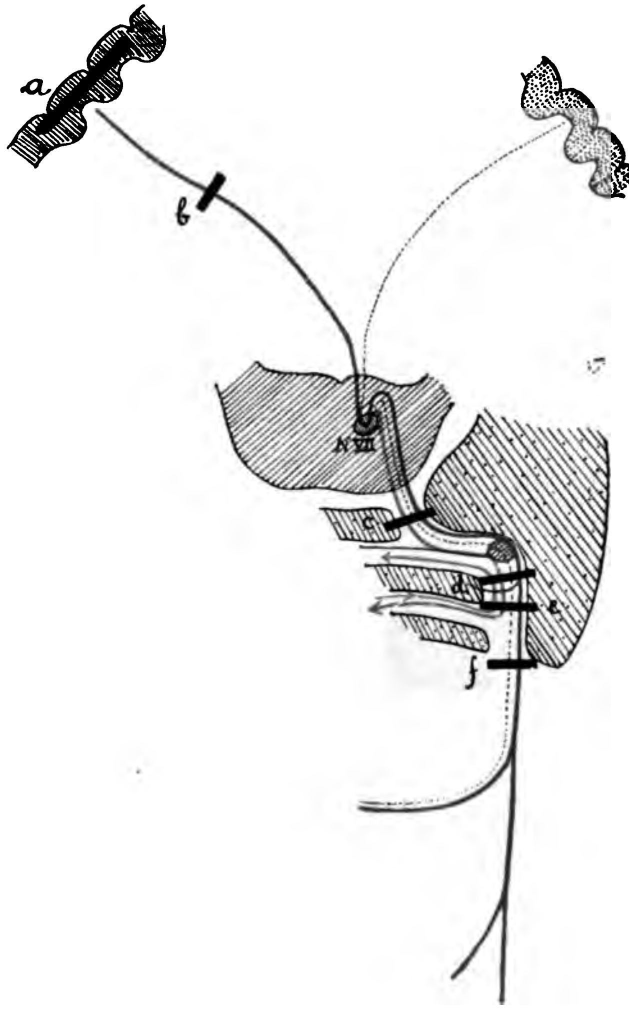


FIG. 10.—Diagram of the principal surgical disturbances of vision. Red and blue = visual fibres. Black = pupillary fibres. III is placed opposite oculomotor nucleus.

The presence of these defects, therefore, excludes a cortical lesion, but their absence only excludes such a lesion when the upper as well as the lower branches of the facial are simultaneously affected.



a. Cortical lesion (trauma, tumour, cyst, abscess). Crossed total paralysis of facial distribution to lower region of face; partial paralysis of upper region of face. Taste and salivary fibres normal. Corneal reflex retained. Stapedius not paralysed. No reaction of degeneration.

b. Lesion of internal capsule (causes as above). Same symptoms, but movements of expression retained.

c. Lesion at exit of 7th N. root (fractured skull). Paralysis of all motor, lachrymal and salivary fibres on same side. Stapedius paralysed (hyperacusis). Corneal reflex lost. Taste fibres unaffected. Expression and associated movements paralysed. R.A. more or less pronounced.

d. Lesion in petrous bone, below geniculate ganglion (fractured skull, mastoid osteitis). Complete motor paralysis on same side. Hyperacusis. Salivary and taste fibres paralysed, lachrymation normal. Expression and associated movements paralysed. Corneal reflex lost. R.A. as above.

e. Lesion below offshoot of N. to stapedius (same causes). Same symptoms, except that stapedius is not paralysed.

f. Lesion at stylo-mastoid foramen (trauma, new growths). Same symptoms, but taste and salivary fibres unaffected.

FIG. 11.—Diagram of injuries to facial nerve. Red = motor fibres. Green = salivary fibres. Blue = taste fibres.

These delicate investigations demand a good deal of time, and also require a due amount of intelligence on the part of the patient. It is only exceptionally, as in the case of growths, and the late results of injury, that the requisite time is devoted to the examination; but if we have to decide quickly, whether and where we should trephine an *paralyses are also present, pointing to injury of the petrous bone. If there be paralysis of the limbs on the side opposite to the facial palsy, the lesion is at the base, in the neighbourhood of the pons, or there may be two separate foci of disease.*

(4) The **auditory nerve** also possesses many points of surgical interest.

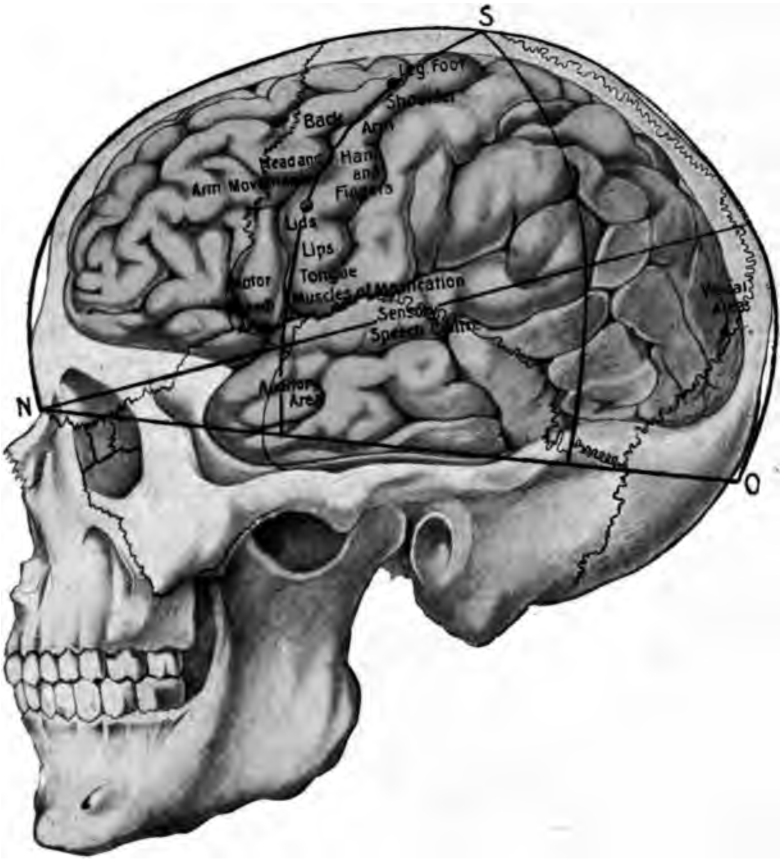


FIG. 13.—Determination of cortical centres after Kocher. Centres filled in partially after Krause.

(*a*) Disturbance of the function of the cochlear nerve—which is the essential nerve of hearing—may be due to pathological changes at the base of the skull or at the base of the brain—injury, inflammation or tumour. We are able to decide, by means of Rinne's and of Weber's tests, whether the nerve itself or the sound-conducting apparatus is involved. If Rinne's test is positive, *i.e.*, if air conduction is better than bone conduction, and if Weber's test shows that the note of the tuning-fork placed on the vertex appears louder in the injured ear than in the other one, we may assume that the disease is in the nerve structures. We cannot, however, tell whether the nerve itself is affected in its course, or the end organ or the cochlea. Even the loss of high-pitched notes (Galton's whistle) only indicates that the damage is in the nerve apparatus, but gives no information regarding its position. There is always some uncertainty as to the significance of auditory disturbances. A definite significance is only possible if the patient's hearing was normal before the onset of the disease from which he is suffering, but in many cases this cannot be ascertained.

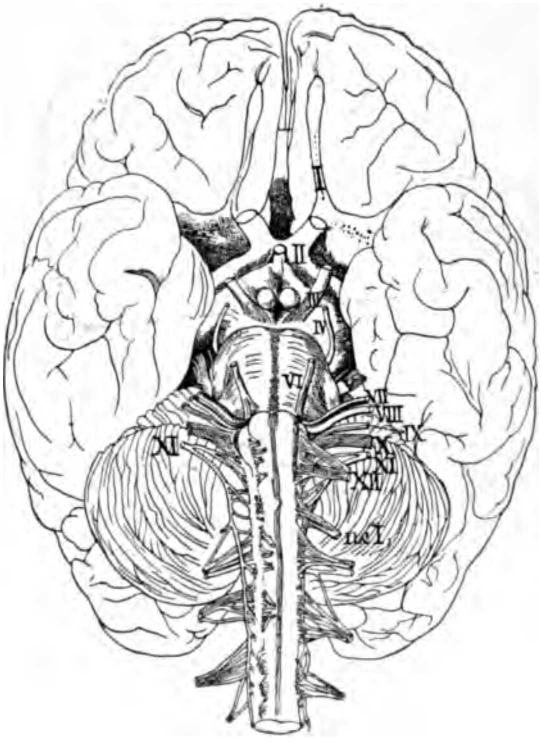


FIG. 14.—Base of brain (adapted from Henle).

(*b*) The *vestibular nerve* is of more importance, surgically, although damage to it cannot be distinguished from damage to the end-organ (semicircular canals)—or, rather, can only be distinguished from it indirectly. As the vestibular nerve is responsible for maintaining the equilibrium, the characteristic symptom of its injury is loss of equilibrium, appreciated by the patient as giddiness — indeed, as rotatory giddiness. But as the fibres of the vestibular nerve terminate in the cortex of the cerebellum, and especially in the vermiform process, it follows that injuries or diseases of the cerebellum produce the same symptoms as giddiness. According to English authorities, the difference between central and peripheral disturbances of the equilibrium apparatus is to be found in the fact, that when the disturbance is peripheral, external objects appear to the patient to be rotating from the healthy to the diseased side, whereas in central disturbances the rotation is from the diseased to the healthy side.

(5) We will not discuss the special circumstances attending those

cranial nerves which are not referred to here, because all the important diagnostic points have been mentioned in their appropriate place.

(6) When there is paralysis of other movements, especially in the *extremities*, the main surgical interest refers to the question whether the lesion is in the cortex or at the base. This must be decided by observing what concomitant symptoms are present, as previously indicated. There is not usually any difficulty in this, because a facial palsy on the same side will point to a cortical lesion, whereas opposite paralysis of the facial, trigeminal, abducens or auditory nerves will point either to a basal lesion or to two separate foci of disease.

Figs. 12 and 13 represent all the points hitherto established.

The more circumscribed the paralysis, the more likely is it to be due to a cortical lesion. Monoplegia of one arm indicates a focus of disease in the cortex, complete hemiplegia indicates damage to the internal capsule or a cerebral peduncle.

(7) **Aphasia** can be diagnosed by the well-known general rules. Its surgical significance is, however, small, whether we adopt the usual view of a definite cortical centre, or attribute the so-called cortical motor aphasia to disease of subcortical tracts.

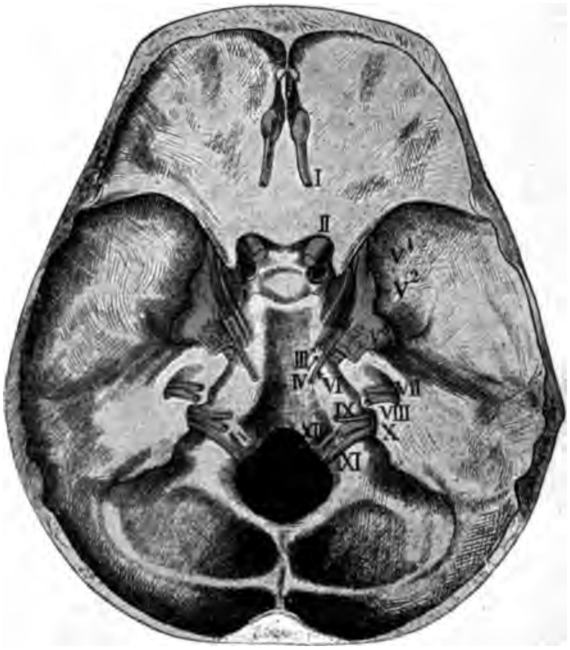


FIG. 15.—Nerves at the base of the skull (adapted from Henle).

v. Monakow has recently endeavoured to explain certain conditions which do not accord with the usual scheme, by assuming that one cortical area may exert an inhibiting effect on another (Diaschisis).

The restoration of the power of speech after destruction of the corresponding cortical convolution on the left side, may, on this theory, not be due to the corresponding area on the right side gradually taking up this duty, but may be explained by the cessation of the diaschisis.

It is important to distinguish between anarthria and aphasia. *Anarthria* means "word mutilation," the result of damage to the peripheral speech apparatus (disturbance of function in the vicinity of the nuclei and the emerging roots). *Aphasia*, on the other hand, means a derangement in the power of "word construction" in the region of the cortex and the subcortical association fibres. Anarthria points to tumours in the neighbourhood of the pons and the medulla oblongata, whereas aphasia indicates damage to the cortical centres and the subcortical fibres. It is easy enough to differentiate in the extreme cases, but difficulty arises in the mild cases, because, in slight cortical and subcortical changes, especially when the interruption of the speech fibres is incomplete, disturbances arise which are hardly distinguishable from nuclear and peripheral anarthria. A correct diagnosis can only be made in such cases by taking other symptoms into consideration (those arising from the pons, medulla, or other cortical symptoms). It should be added that the speech centre is usually on the right side in left-handed people.

The study of aphasia has established the following points in regard to the surgical aspect of local diagnosis :—

(1) Definite pure anarthria indicates a lesion in the cerebral peduncle, pons, or medulla oblongata.

(2) If the differentiation between anarthria and aphasia is not quite clear, the remaining physical conditions will enable the diagnosis to be made.

(3) Definite motor aphasia indicates a lesion in Broca's convolution (inferior frontal convolution).

(4) Definite sensory aphasia indicates a lesion in Wernicke's convolution (superior temporal convolution).

Numerous methods and instruments have been devised for *defining the cortical areas on the surface of the skull.* We shall here only refer to the useful and practical methods of Kocher and Kroenlein. The whole procedure is greatly simplified by employing the special craniometer invented for the purpose; but a tape measure and a blue pencil will do quite well. The most important thing is to define the *anterior central convolution*, wherein the chief motor centres lie. This convolution lies between the central and precentral sulcus. Kocher's method depends upon the simple and certain definition of this latter landmark.

The *base line* is first drawn, by placing the tape measure horizontally just above the concha, and through the glabella and external occipital protuberance. The lower border of the tape measure, fixed in this position, is marked off with the blue pencil (figs. 16 and 17). The *sagittal meridian* is defined in a similar manner, by placing the tape over the vertex, from the glabella to the external occipital protuberance (N, S, O., fig. 13). The length of this line is measured (in adults, about 35 cm.), and its midpoint is marked on the vertex, *i.e.*, 17½ cm. from either end (S, point of vertex). A second meridian must now be defined, with the craniometer, which should meet the

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sagittal meridian at an angle of 60°. This meridian can be ascertained, within 2 or 3 degrees of perfect accuracy, in the following manner. One half of the base line is divided into three equal parts, and the tape is placed with one end on the hinder point of the first division *c*, and the other end on the point of the vertex S. (In adults the point *c* is 9 to 9½ cm. behind the glabella.) The meridian so obtained defines the position of the *precentral sulcus* (Pr) behind which the chief centres are to be sought for.[1] If this meridian be divided into three equal parts, the lower points of the two upper divisions (*a* and *b*) will correspond to the hinder extremity of the two frontal sulci. The upper third will indicate the leg centres, the middle third the arm centres, and the lower third the face centres. In order to define the fissure of Sylvius the tape is applied from the root of the nose to the upper end of the squamous portion of the temporal bone, at the beginning of the lamboid suture. The portion of this line behind the precentral sulcus, corresponds in its whole extent to the Sylvian fissure (fig. 13).

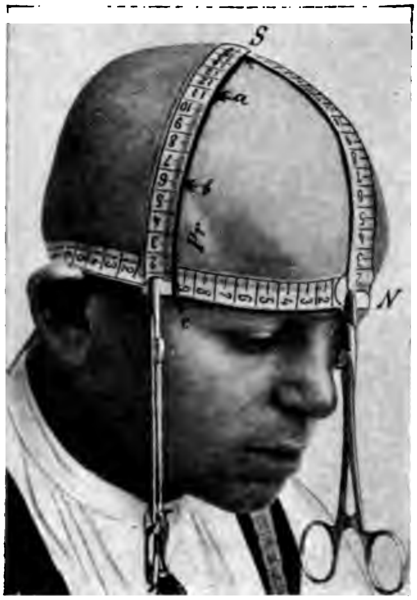

FIG. 16.—Simplified method of defining precentral sulcus.

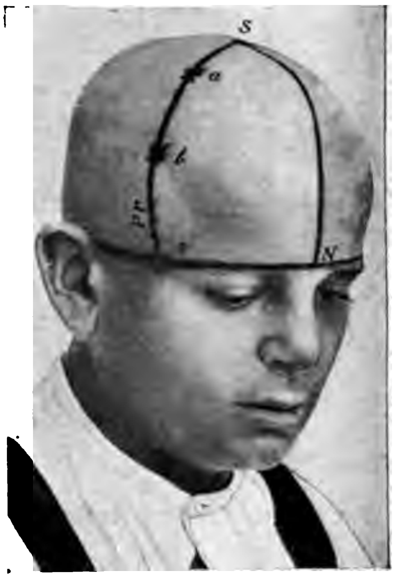

FIG. 17.—Simplified method of defining precentral sulcus.

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[1] Kocher's precentral line obtained by angle measurement lies farther behind. It can be obtained by moving the point *c* ½ cm. posteriorly. There will then be a difference of 1½ to 2 mm. in the point marking the hinder extremity of the superior frontal sulcus, and a difference of 3 to 4 mm. in the point marking hinder extremity of the inferior frontal sulcus. But I have frequently demonstrated on the cadaver that, as all these methods possess inevitable errors, this difference is of no essential consequence.

Other topographical areas ascertained by means of these fundamental lines are indicated in figs. 12 and 13, which are partially based on the latest work of Krause.

Kroenlein's method of measurement is particularly useful in the search for a hæmatoma of the middle meningeal artery, or for abscesses of brain, due to ear disease.

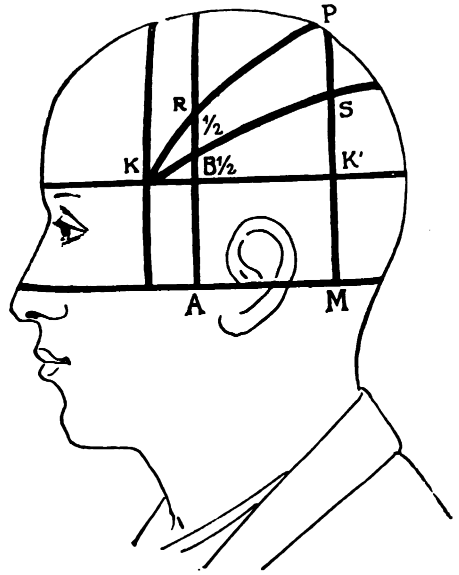


Fig. 18.—Craniometry (after Kroenlein).

The *base line* is determined by means of tape-measure and blue pencil; the tape being placed at the level of the infra-orbital margin and the external auditory meatus. A *superior horizontal* line is marked off, parallel with this at the level of the supra-orbital margin. Three perpendicular lines are then drawn in the following positions: (1) *anterior*, through the middle of the zygomatic arch; (2) *middle*, through the condyle of the lower jaw; (3) *posterior*, through the hindmost point of the base of the mastoid process. By joining the point where the anterior perpendicular line intersects the superior horizontal, with the point at which the posterior perpendicular intersects the line of the vertex of the head, we then get a line which corresponds to the fissure of Rolando. By bisecting the angle made between this line and the superior horizontal, we obtain the direction of the fissure of Sylvius. The points K and K' (fig. 18) indicate Kroenlein's landmarks for trephining in cases of hæmatoma of the middle meningeal artery. The square A B K' M is the area suggested by Bergmann for searching for cerebral abscess of aural origin.

CHAPTER VII.

THE SURGERY OF EXOPHTHALMOS.

A STARING eye always calls for an answer to the question whether it is a real enlargement of the organ, a *buphthalmos*, or a protrusion of the organ—an *exophthalmos.* The former has a certain amount of surgical interest when it is caused by a tumour, a sarcoma of the choroid or glioma of the retina. But as these growths concern the speciality of ophthalmology, which, however, gains very little credit from them, we shall here limit ourselves to the discussion of *exophthalmos.*

If a *bilateral* exophthalmos has come on *gradually* during a course of months or even years, and if it is accompanied by tachycardia and tremors, we may at once diagnose *Graves's disease,* which will be fully discussed in the section on Swellings of the Neck.

If a *unilateral* exophthalmos has come on in the course of weeks or months, and is accompanied by double vision, the cause is either a tumour developing within the orbit, or one invading it from without, starting from the base of the skull or the upper jaw—usually a sarcoma. If the symptoms have been slow in manifesting themselves, we should think of an innocent growth, viz., an ivory osteoma of the orbital bones, or a fibroma of the base of the skull penetrating the orbit.

A *unilateral exophthalmos* which has come on *gradually* after an *injury,* and which is accompanied by pulsation of the eyeball, and by a buzzing noise synchronous with the carotid pulse, heard with the stethoscope in the temporal region, represents the clinical picture of what may be briefly called a **pulsating exophthalmos**. This is caused by a rupture of the internal carotid artery into the cavernous sinus, and it is impossible to confuse this condition with any other. But if confirmatory signs are required, these may be found in the dilatation of all the ocular and conjunctival vessels, in the congested disc, in the progressive loss of sight, and in the gradual onset of paralysis of the sixth nerve.

Any injury involving the neighbourhood of the cavernous sinus may produce this condition. A fractured base is the most frequent cause, but stabbing and shooting injuries are occasionally responsible. The same clinical picture sometimes arises quite spontaneously, without any injury, and is then due to *aneurysm of the ophthalmic artery,* or a *cavernous angioma of the orbit,* in which latter the pulsation is not well marked. Finally, exophthalmos pulsans has also been noted as a consequence of venous congestion, due to severe pressure during birth.

In determining the etiology, it is important to realize that the symptoms may not appear for months or even years after the injury.

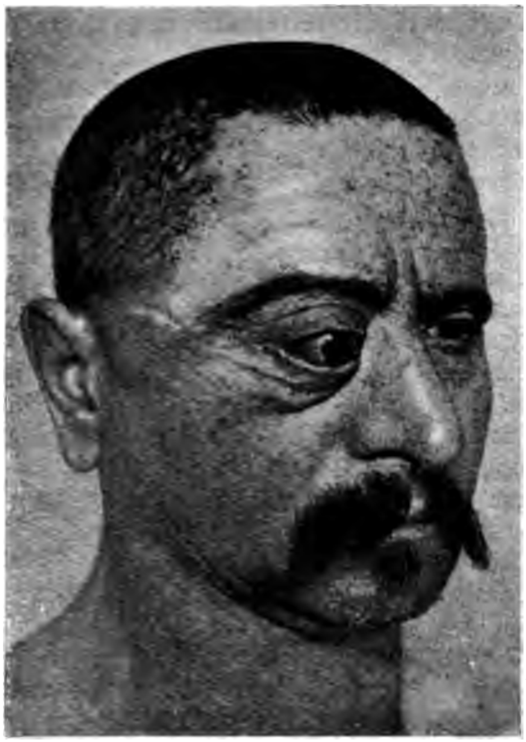


FIG. 19.—Exophthalmos due to a retro-bulbar tumour.

An *acute* exophthalmos, either *unilateral or bilateral*, generally depends upon a retrobulbar hæmorrhage, usually the result of a fractured base, but not necessarily so. The presence of suffused lids, either immediately or coming on after a day or two, is a further indication of the cause at work.

If all these causes are absent, there remain only some inflammatory process to be considered, either a retrobulbar *thrombosis* or *abscess*. The differential diagnosis of these two conditions will be detailed later on.

CHAPTER VIII.

ACUTE INFLAMMATORY PROCESSES ON THE SKULL.

APART from **erysipelas**, which is usually a secondary affection of the scalp, and which is peculiar in the absence of reddening of the skin, the acute inflammations concern the glands or the bones. **Inflamed glands** will be found behind the ear, or in the posterior cervical region. The portal of infection is generally a slight superficial abrasion or a moist eczema, and there is never any difficulty about the diagnosis. Infections of the *bones* are, however, not quite so simple. If the inflammatory process is situated behind the ear, a purulent otitis should be sought for, and it will generally be found. It very rarely happens that the infection misses the middle ear, and settles forthwith in the cells of the mastoid process. In these rare cases, however, it will be possible to trace a previous sore throat or influenza, &c. If the inflammatory process is located elsewhere, we must resort to the diagnosis of **acute periostitis and osteomyelitis** of the skull. This diagnosis is easily made if the infection of the cranial bones represents a metastasis from a distant focus of osteomyelitis elsewhere. But if this is not the case we must recognize in these symptoms a **primary osteomyelitis** of the skull, although this is a rare condition. It is important to make an early diagnosis, because if operation is delayed there is the danger resulting from the suppuration advancing to the inner table of the skull bone.

CHAPTER IX.

SWELLINGS ON THE HEAD.

OUR first inquiry concerning any swelling on the head should be to ascertain whether it is congenital. If it be so, the diagnosis is either: (1) *Cerebral hernia*, (2) *Hernia of the meninges*, (3) *Angioma*, or (4) *Dermoid*. Other congenital swellings are very rare, and are generally connected with a concealed cephalocele.

A.—CONGENITAL SWELLINGS OF THE HEAD.

(1) Cerebral herniæ are situated in the middle line, mostly above the *nose*, or on the *occiput*.

Cerebral herniæ at the base are very rare and do not possess any surgical interest.

Cerebral herniæ on the forehead form moderately large, flat, semicircular, or sometimes irregular swellings, and they chiefly contain brain substance; hence their name of *encephaloceles*. Cerebral herniæ at the *back of the neck* are much larger. They contain either a dilated posterior cornu surrounded by a thin layer of brain substance, or a very dilated retiform arachnoideal space with a plug of brain substance at its base (*encephalomeningocele*). A simple protrusion of the meninges, a pure *meningocele*, is a much rarer condition.

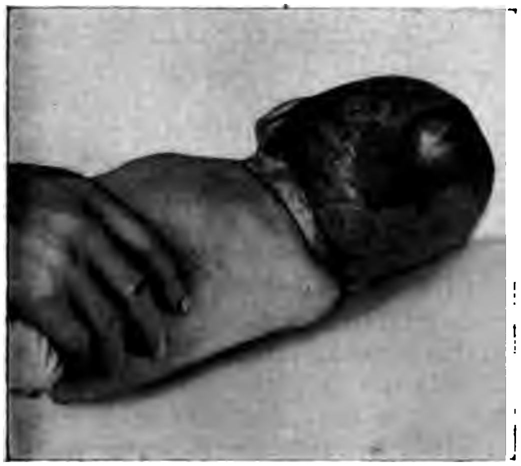


FIG. 20.—Superior occipital encephalomeningocele (with a vascular nævus in the region of the cerebral hernia).

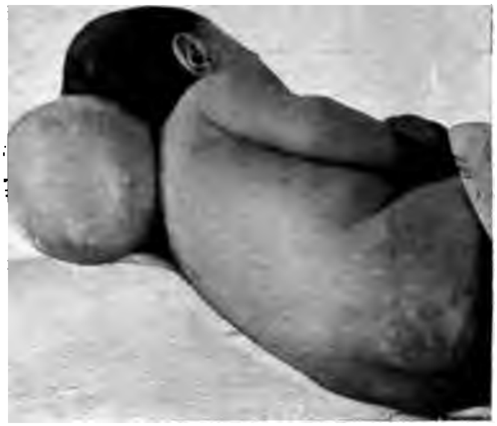


FIG. 21.—Inferior occipital encephalomeningocele.

Many cases which were previously regarded as pure meningoceles are really encephalomeningoceles of one of the previously mentioned types. Sometimes a microscopic examination is necessary before the real nature of the deformity can be determined.

Superior (fig. 20) and *inferior occipital cerebral hernia* (fig. 21) are distinguished according to their position, *above* or *below* the occipital protuberance.

Whenever these swellings become *more tense on crying*, it signifies a communication with the interior of the skull. The same applies to *pulsation*, which, however, is often absent in the case of cystic hernia of the brain. *Displacement* on steady pressure occurs with meningoceles and cystic encephaloceles. This depends upon the extent of the connection with the interior of the skull, and it is obvious that in large swellings the displacement is very imperfect. It is not always possible to detect the hernial aperture, *i.e.*, the gap in the skull, which may be quite small, even when the swelling is large. We sometimes find, especially in large meningoceles, that an incipient *hydrocephalus* is present, which it is most important to recognize, because this complication excludes the possibility of a successful operation.

These congenital swellings must be diagnosed from :—

(*a*) Hæmorrhagic cyst of a sinus, the so-called *Sinus pericranius.* This swelling emanates from within the skull, it is situated in the middle line, generally on the occiput, but it contains blood instead of cerebrospinal fluid. If the swelling is large, and the overlying skin is thin, the dark blue blood will be visible through it ; but if the swelling is small, and the overlying skin has not been thinned out, we shall get no assistance from the colour. But in these cases, the swellings are very easily emptied by pressure, because the cerebral sinuses have many more outlets than the sabarachnoid spaces.

(*b*) *Cavernous angioma* which has formed a secondary communication with the longitudinal sinus, as seen particularly at the anterior fontanelle. It is easier to displace the contents of this swelling than a cerebral hernia, and the cavernous structure of the swelling can be recognized through the skin. Furthermore, cephaloceles at the anterior fontanelle are so rare that their existence has been recently denied.

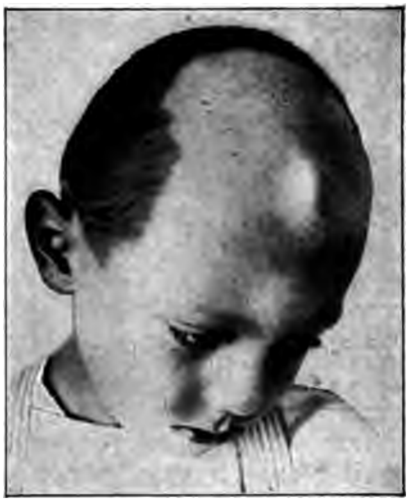


Fig. 22.—Dermoid cyst, situated in a gap of the skull.

(*c*) *Dermoid.*—In this case there are no signs of communication with the interior of the skull, unless the dermoid is situated in a gap of the skull and presses directly on the dura mater, as in the patient depicted in fig. 22. But even in this last type of case the symptoms noted are very much less definite than in cerebral hernia.

If a child presents a pulsating, displaceable, dome-shaped swelling of the integument of the head, not necessarily in the middle line, accompanied by more or less evident defect of the bone, it must be regarded as a **spurious meningocele** due to some previous injury—a **traumatic cephalo-hydrocele.** This may be caused when a child has sustained an injury to the skull with contusion of the brain, and a communication has formed between the subcutaneous tissue and one of the cerebral ventricles. The condition is really a traumatic porencephaly, complicated with an effusion of cerebrospinal fluid under the skin. The injury may have been inflicted at birth, with the forceps, when it will be regarded as congenital, or it may have been caused by a blow or a fall; not unfrequently by dropping the child. If the child is rickety the defect in the skull may gradually increase, and therewith also the effusion of the fluid under the skin. In cases of forceps injury we have seen the condition subside completely. The swelling gradually disappears in the course of a year, and is only apparent if the head is in a dependent position, and eventually the aperture is entirely filled in by dense fibrous tissue. Its characteristics are, therefore, quite definite in every stage. If the injury has involved the motor area, an incurable spastic hemiparesis will follow, corresponding completely to the clinical picture of a cerebral infantile paralysis. The examination of the opposite half of the head should, therefore, never be neglected in this variety of paralysis.

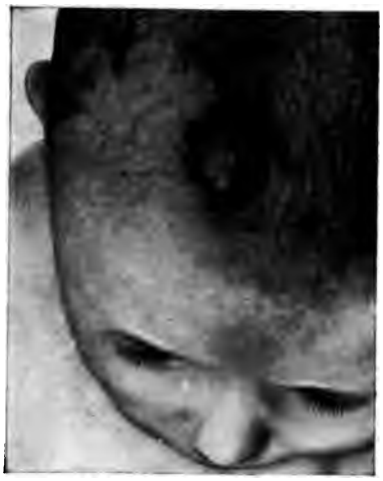


FIG. 23.—Mulberry-shaped angioma in an infant.

Traumatic meningoceles not communicating with the lateral ventricle are very rarely met with. They are small and are only replaceable with difficulty.

(2) We have mentioned **angioma** as the second variety of congenital swelling. All grades of this variety exist, from teleangiectatic stains, which scarcely project above the skin, to large cavernous angiomata. They may occur anywhere on the skull, and the fact that they are freely movable over the bone shows that they are situated in the skin. Their finely nodular surface (fig. 23) and the hue of the blood visible through them as a red or blue colour, permits of the framing of an immediate diagnosis. We have already seen that a cavernous angioma can penetrate deeply and open into a sinus.

(3) **Dermoids** are situated under the skin, generally in the orbital region, more rarely at the back of the neck, but very rarely on the vertex. They are but slightly movable over the bone, and are often

situated in a depression thereof, occasionally even infiltrating it. They can frequently be detected in children, but as a rule they only develop in later life to a size which is noticeable (fig. 30).

(4) **Atheromata** (Sebaceous cysts) should also be mentioned in this connection. Although these probably develop from some congenital antecedents they are not usually evident until after the age of 20 or more, appearing anywhere on the skull, sometimes even in crops of dozens. They vary in size from a lentil to a child's head (figs. 24 and 25). Their position and their freedom from the subjacent bone distinguish them from dermoids. The frequently observed family predisposition proves their congenital character.

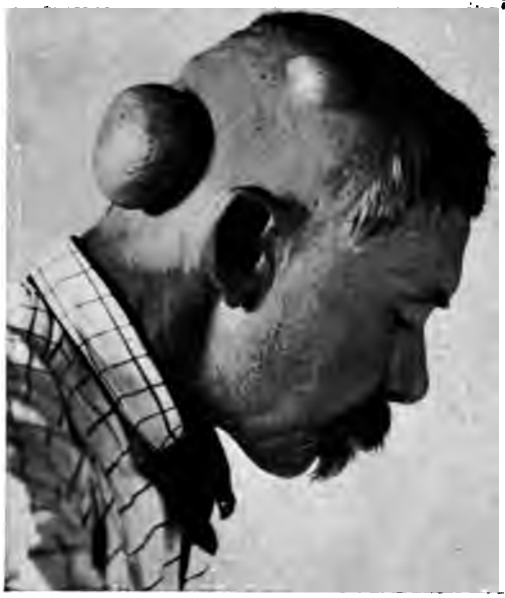


FIG. 24.—Sebaceous cyst on scalp.

Sometimes these cysts open spontaneously, leaving a sinus from which fatty material discharges itself from time to time. As they usually become affected, suppuration also occurs. Their malignant degeneration is referred to later on.

B.—ACQUIRED SWELLINGS OF THE HEAD.

Sebaceous cysts lead us to swellings of the head and tumour-like formations, which develop in later life. We have here to distinguish between innocent and malignant structures.

(1) *Innocent Acquired Swellings.*

These are soon disposed of, because they include only **osteoma** (fig. 26), often of an ivory consistence, and **soft fibroma** of the skin. The latter is usually but one of several generalized fibromata, and therefore the diagnosis can be made at first sight.

Sometimes a few apparently soft harmless warts may, after existing for years, lead to a fatal result, with the severe symptoms of Recklinghausen's disease.

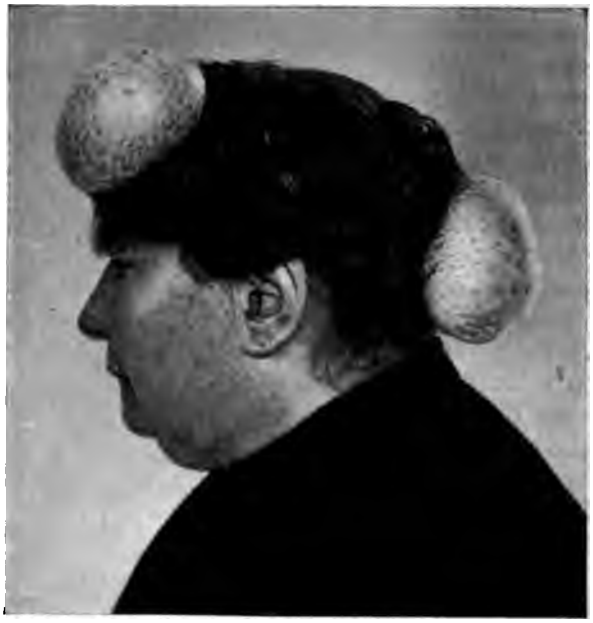


FIG. 25.—Giant sebaceous cysts.

(2) *Malignant Tumours of the Head and Chronic Inflammatory Swellings.*

(*a*) Swellings which are *closely connected with the bone* may be of the nature of *gumma, tubercle, sarcoma,* or *metastatic tumour* of any kind.

Gummata, which never reach any significant size, are generally multiple, and when recognizable as tumours are usually soft in the centre. They begin as hard thickenings of the periosteum, which may be distinctly painful on pressure, thus contrasting with the usual painlessness of tertiary syphilitic lesions. When ulcerated, their specific character is attested by the yellowish, fatty base of the ulcer, by the superficial necrosis of the bone, by the irregular ivory-like movable sequestrum, and by the new deposit of bone round about. If no history of syphilis is forthcoming, traces of its attack will probably be found in the form of scars and of irregularities on the bones. In a doubtful case there will always be time to try the effect of potassium iodide and to apply the Wassermann serum test.

Tubercle of the skull, an affection more frequent in children than in adults, is also often multiple in character. A focus of tubercle undergoes softening much earlier than a gumma, and as the disease extends a cold abscess is formed. These two points are in themselves sufficient to distinguish the two conditions, especially as in tubercle there are probably other foci of disease, if only tubercular glands. But in no circumstance should a non-acute fluctuating swelling of the skull be incised, unless one is prepared to proceed further forthwith. If the abscess be opened or if it burst spontaneously, the ulcer in the bone is seen to have a sharp edge as if punched out, and if there be a sequestrum it is movable, easily liberated, and there is no new bone formation round about. These characters will clench the diagnosis.

If we are able to exclude both gumma and tubercle, the only condition which remains is a **malignant growth.** As metastases in the skull are not rare, we should first search for a primary focus, especially in the *thyroid gland*. It is well known that even an apparently innocent goitre may give rise to secondary deposits of a malignant character. These may occur anywhere on the skull and may grow deeply into the cranial cavity. In contrast to certain sarcomata, they are remarkably soft to the touch. In the absence, however, of any cause for the growth, it must be regarded as a **primary sarcoma,** which may originate from the periosteum of the skull, or dura mater. A periosteal origin is suggested by the absence of transmitted cerebral pressure, while the early onset of headache and brain symptoms suggests an origin from the dura mater. No definite conclusions can be drawn from the effect of the growth on variations in the intracranial pressure, because the tumour may just as likely grow from

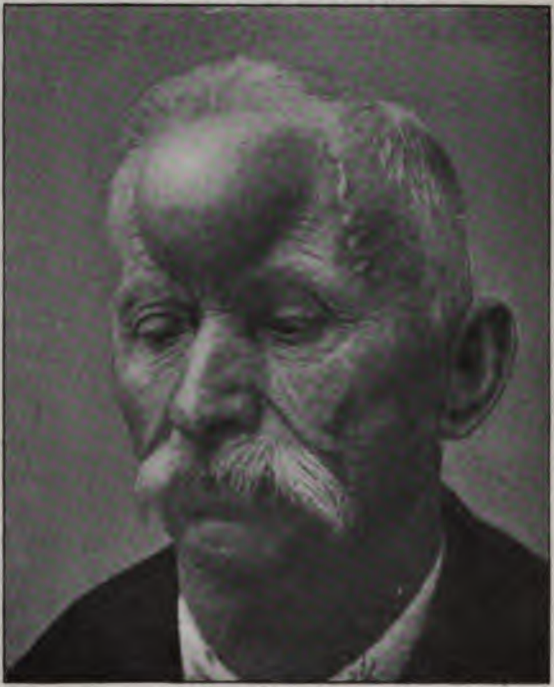


FIG. 26.—Osteoma of frontal bone.

within outwards, as from without inwards. The cessation of headache is very striking when a tumour of intracranial origin has perforated the roof of the skull.

(*b*) In cases of *chronic ulceration* of the skull the problems are somewhat different. If the ulceration has attacked the bone, and the patient states that it was preceded by a tumour-like condition, we must regard it as **gumma of the skull,** especially when the base of the ulcer has a yellowish, fatty appearance. With a similar history, and an ulcer freely movable over the bone, we should consider it to be an open gumma of the skin. But if we are told that the condition was an ulcer from the very beginning, we must think of tertiary syphilis and epithelioma. We recognize the former by its shape, base, and border, apart from the history. If grayish plugs can be pressed out of its border, and if at the same time the well-known predisposing changes are present on the skin, viz., seborrhœa and verruca senilis, there is very little difficulty in arriving at the diagnosis of **seborrhœic epithelioma.** In this instance multiplicity does not contraindicate the diagnosis of primary malignant new growth. Finally, if the patient states that a sebaceous cyst, which he has had for many years has become hard, ulcerated and painful—probably after an injury to it—there can be no doubt that the diagnosis is cancerous degeneration of the cyst (see fig. 27).

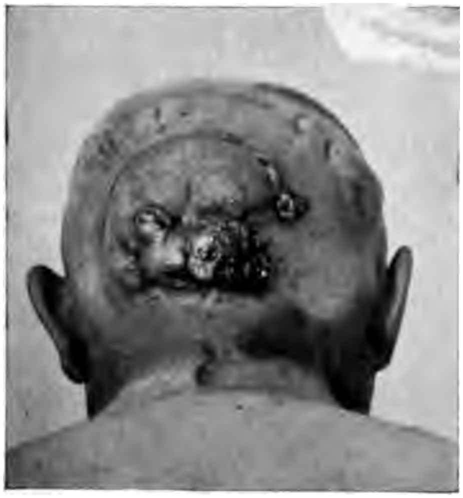


FIG. 27.—Sebaceous cyst of head, undergone cancerous change.

CHAPTER X.

ACUTE INFLAMMATORY DISEASES OF THE FACE.

ALL inflammatory processes about the face rapidly produce severe swelling, because the skin, especially of the lips and cheeks, is richly supplied with blood-vessels, and the subcutaneous tissue of the eyelids is very loose. Wherever the origin of the inflammation may have been, there is a considerable resemblance in the appearance of all these cases. The lips are puffed and protuberant, the cheek is bloated, the eyelids are converted into smooth pads, between which the eyes peep with difficulty, if they are visible at all. If we are confronted with such a case, an immediate diagnosis cannot be made without the previous history and more detailed examination. The possibility of **erysipelas** will suggest itself first. This will be confirmed by the symmetry of the swelling, by the deep redness of the skin and the sharp limitation both of the swelling and the redness. We must not look for this limitation in the neighbourhood of the eyelids, because they swell up at once to their full extent, owing to their anatomical characteristics; the margin will be on the cheek or forehead; the patient usually states that the swelling began on the nose. If he has already had previous attacks, which is very likely, he will supply the diagnosis himself and will only want to know whether any improved method of treatment is available. A careful examination of the swelling will show that it is situated in the skin, and that the deeper tissues are free. But if erysipelas is excluded, the diagnosis usually lies between one of the following conditions—*Furuncle of the upper lip, periostitis of the upper jaw, acute inflammation of the maxillary sinus,* or the *frontal sinus, inflammation of the lachrymal sac, inflammation within the orbit,* and finally, accidental infections of various kinds.

In regard to **furuncle of the upper lip,** which, at times, infects the facial veins and leads to pyæmia, the original site of infection must be ascertained from the patient. It is easy to overlook a furuncle, which is not usually large. Occasionally a considerable portion of the upper lip is occupied by foci of pus.

Acute **periostitis of the upper jaw,** and an acute exacerbation of **inflammation of the maxillary antrum,** at first sight, appear to have the same symptoms. Besides the swelling, we are struck in both with sensitiveness on pressure over the canine fossa and the vestibule of the mouth, the superficial soft parts not being affected. The difference will usually be evident from the history. In ordinary periostitis there will have been previous toothache, and we shall probably find a carious tooth or one sensitive to pressure. In a recent sinusitis, a history of a preceding cold or influenza will often be elicited. If the condition is due to an acute exacerbation of an old sinus inflammation, careful inquiry will reveal the typical history of all sinus catarrhs with periods when they become much worse and give rise to profuse discharge of sanious and often foul-smelling pus. Objective examination will frequently afford points of differentiation. In *dental periostitis* the swelling of the soft parts—including the gums—is most prominent, and the sensitiveness to pressure of the upper jaw concerns mainly the alveolar process; but in *acute sinusitis* there is less swelling of the soft parts. In this condition the bone is sensitive to pressure as far as the infra-orbital margin, and in addition there is often a neuralgia affecting the cutaneous branches of the infra-orbital nerve. The nasal mucous membrane on the diseased side is œdematous, and nasal respiration is obstructed. In *chronic sinusitis* there is generally hypertrophy of the mucous membrane over the middle turbinated bone, and polypi are also present. If the patient does not happen to be suffering from retention of the discharge at the time of the examinations, we may be able to detect pus escaping from beneath the middle turbinated bone if the examinations are repeated often enough. Finally, diagnosis may be facilitated by puncturing the sinus from below the nasal duct.

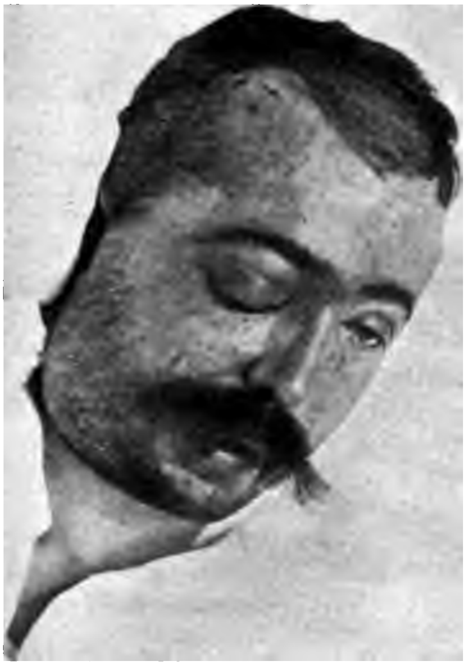


FIG. 28.—Thrombosis of cavernous sinus and retro-bulbar phlegmon following injury to frontal bone.

If the inner angle of the eye, which is generally swollen up and very painful, is the centre of the inflammatory disturbance, the condition originates from the lachrymal sac, and a phlegmonous **Dacrocystitis** exists. The tears run over the lid, showing that the lachrymal canal is blocked, and the patients, who usually have had previous attacks and are well posted in the details of their cases, will often relate their experiences of probing the tear duct.

These inflammatory processes have frequently been traced to a streptothrix infection.

Inflammation starting from the **frontal sinus** is recognized by the forehead and upper lid being chiefly involved. Its previous history resembles that of inflammation of the maxillary sinus.

Mild cases of frontal sinusitis are often mistaken for supra-orbital neuralgia. It is quite true that secondary inflammation of the nerve may occur, but it is important to recognize the original and causative factor. Pressure on the wall of the sinus *near* the nerve generally suffices for this.

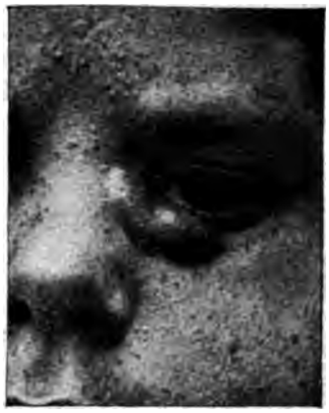


FIG. 29.—Tuberculosis of the lachrymal sac.

As we are dealing with the bony margin of the orbit, we may refer to a not uncommon disease in children, which sometimes presents itself as an acute inflammation—*i.e.*, **tubercle of the superior maxilla,** affecting the lower margin of the orbit. This leads to sinus formation, and as there is invariably some secondary infection, extensive swelling of the soft parts occurs if secretion is retained. Tubercle of the lachrymal sac should be mentioned here, a typical illustration of which is seen in fig. 29.

Inflammatory diseases of the *orbital contents* present a uniformly similar picture, because the swelling is more or less sharply circumscribed by the conjunctiva and the lids, and because the globe of the eye is usually protruded. The only exception to this last symptom is in **inflammation of the lachrymal glands,** which is recognized by the situation of the swelling, chiefly on the outer side of the upper lid and the adjoining part of the forehead. If the globe is protruded and both lids are puffed like pillows, the diagnosis can only be between a **retrobulbar abscess** and a **venous thrombosis in the retrobulbar fatty tissue, or in the cavernous sinus.** It is not easy to distinguish between these two processes, because both the symptoms and the etiology are alike, the cause generally being some focus of inflammation on the face or the bony structure, and because both conditions may co-exist (see case in fig. 28). Facial erysipelas, furuncle of the face, ostitis and periostitis of the upper jaw are mainly responsible for these conditions.

If the symptoms are on *both sides,* they must be due to *thrombosis of both sinus cavernosi.* Surgical intervention has, unfortunately, failed hitherto in these cases.

A little boy who was suffering from a persistent suppurative periostitis of the jaw had a lower molar extracted by a quack—the same thing may have happened if the operator had been a dentist or a doctor. Immediately afterwards, the lids of both eyes began to swell, the globes protruded, rigors occurred, and the temperature rose to 102°—104° F., the pulse failed, consciousness was lost, and death ensued a few days after the onset of the symptoms, as is usual with septic thrombosis of the sinus cavernosi.

If the disease is *unilateral* we must seek to differentiate as quickly as possible between thrombosis and abscess, in order to institute appropriate treatment. As long as both lids are equally swollen without any circumscribed redness, and in the absence of severe pyrexia, we may regard the condition as retro-orbital thrombosis without purulent softening, and we are justified in waiting. But if one lid, especially the upper, swells up more and becomes redder and painful, it means that there is pus deeply situated and we must interfere.

The following case is very instructive in this connection. A patient who had been bitten by a friend in the left upper lid, was brought into the hospital suffering from erysipelas. The disease spread over the entire head, and caused several subcutaneous abscesses. After these had healed, swelling of the lids suddenly started again on the side which had been bitten, and exophthalmos followed. The swelling was equally distributed over both lids, but there was no very striking puffiness at any one place. The temperature remained normal. The history suggested an abscess, but the regularity of the swelling and the absence of fever nevertheless made it probable that the condition was thrombosis. We awaited developments, ready for immediate operation. In a few days symptoms began to subside, and finally vanished completely without any complication. The diagnosis of thrombosis was evidently correct, and an untimely operation would only have done damage.

If the thrombosis of the cavernous sinus is indicated by severe general symptoms, by high temperature, cerebral involvement, and a narrow fixed pupil, which does not react to light, the case is quite hopeless as far as treatment is concerned, even if the condition be unilateral.

Acute parotitis remains to be mentioned among the acute inflammatory swellings of the face. The malady is immediately recognized by the situation of the swelling in front of the ear and by its extension to the tissues behind the ear lobule. If the temperature is high and both sides are quickly involved, the diagnosis is *epidemic parotitis*, recognized by the stupid frog-like expression which the features acquire when the disease extends in this manner. Orchitis, which is a frequent complication in some epidemics, affords an unwelcome confirmation of the diagnosis. The *unilateral parotitis* which follows infectious diseases—typhoid fever, erysipelas, appendicitis, &c.—is quite different in character, and usually breaks down into an abscess. Finally, there is the acute swelling of the parotid gland due to obstruction of Steno's duct by *salivary calculi.* This condition is characterized by the frequency of relapses.

CHAPTER XI.

TUMOURS AND ULCERS OF THE FACE.

THE first matter to determine in regard to any swelling of the face is whether it originates in the skin or the deeper tissues. If the skin is movable over the growth, the latter has a deep origin ; but if it is not possible to pick up the skin free from the growth, it is obviously situated within the skin, as long as the growth and the skin are together movable over the subjacent tissue. If the skin, tumour, and underlying tissues are welded together into one immovable mass, it means that either the tumour has penetrated into the skin from the deeper tissues, or has invaded these tissues from the skin. Experience shows that the former contingency is the more frequent.

A.—TUMOURS WITH OVERLYING SKIN UNBROKEN.

We limit ourselves to *tumours of the skin*, because we have already considered new growths arising from deeper structures, when discussing diseases of the skull and jaw. **Dermoids** and **sebaceous cysts** need not detain us long. Every beginner recognizes that a semi-globular tumour of the skin is a sebaceous cyst, and he is usually correct, if the growth is really situated *in* the skin. But if the skin can be picked up distinctly free from the tumour then the latter is more deeply placed, and is not a sebaceous cyst, but a subcutaneous **dermoid**. The superior orbital margin is its favourite site. It is important to differentiate between these two, because a dermoid is not so easily shelled out as a sebaceous cyst. A dermoid is frequently seated in a depression of bone, and requires detaching therefrom (fig. 30).

It is hardly necessary to warn against confusing a hernia of the brain, either with a dermoid or a sebaceous cyst. Fig. 31 indicates how easily such a mistake might be made, the sebaceous cyst occupying the position of a frontal cephalocele.

Sebaceous cysts are especially prevalent on the cheeks and the temples, but **lipomata** also occur there, and the differential diagnosis is not always easy.

We need not devote much time to **angiomata** of the various kinds (see fig. 33). Their shape and colour proclaim their diagnosis, and any doubt is dispelled by the fact that they can be dispersed by pressure. A pulsating mass, like earthworms in outline, will be identified, by a beginner even, as a *cirsoid aneurysm.*

Xanthelasma, a flat, pale, yellow growth, often situated symmetrically in the neighbourhood of the lids, is at once recognizable by its colour.

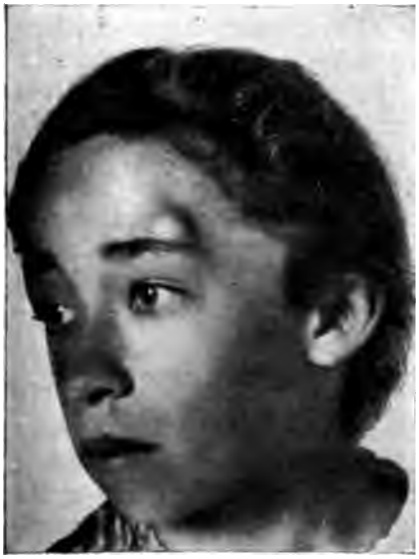


FIG. 30.—Supra-orbital dermoid.

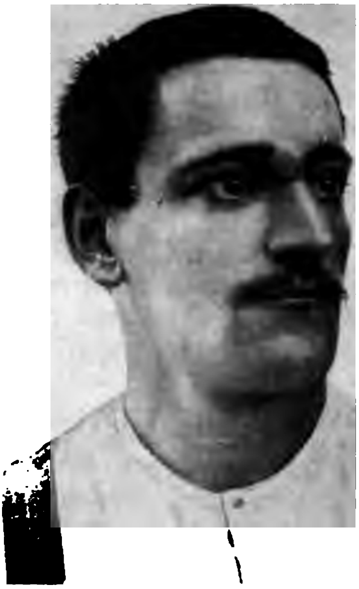


FIG. 31.—Sebaceous cyst at root of nose.

A *calcified epithelioma*, so-called, may be referred to here as a rarity. It appears as a round, flattened, and somewhat nodular tumour, which can be shelled out of the skin quite easily, and is chalky on section. If once seen, it is always recognizable, but the inexperienced cannot be expected to be able to make the diagnosis.

The **cartilaginous pendulous tumours of the skin**, in front of the ear, and the **soft cutaneous warts** so frequent on the face of an old syphilitic subject, are too obvious to detain us. But these soft cutaneous warts acquire something more than cosmetic importance when they become malignant. Rapid growth, an increasing firmness of consistence, and bleeding when lightly touched, are clear proofs of the change.

Angioma and **Lymphangioma** of the ear are not very common, but they are easily recognizable. Lupus in this situation often resembles a tumour (fig. 54), and **hæmatoma of the ear,** which occurs in the insane, is also strikingly similar to a tumour (fig. 32).

B.—ULCERATIVE PROCESSES.

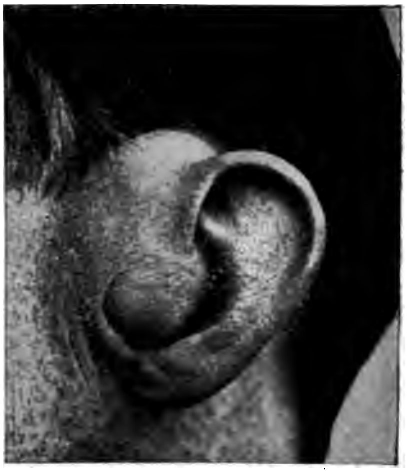


FIG. 32.—Hæmatoma of the ear. (From the Ear Clinic, Basle.)

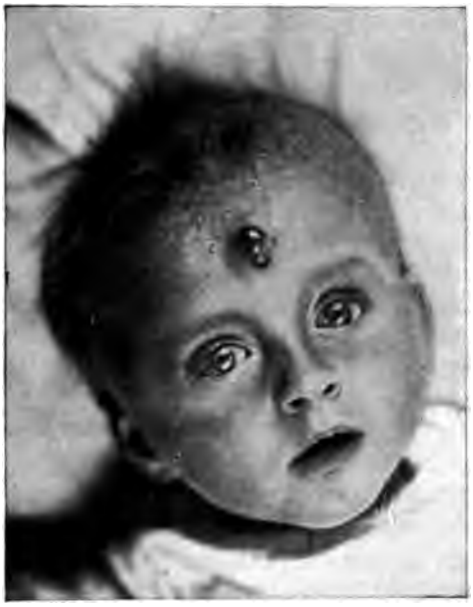


FIG. 33.—Angioma of the forehead.

Greater diagnostic interest attaches to lesions of the skin of the face, which are ulcerated from the beginning, or have broken down after a brief duration.

(1) We shall adopt a topographical order, starting with the **lips.** A deep **chronic ulcer of the lip** is either a **primary sore** or a **cancer** (fig. 36.) The hardness of the base constitutes no distinction, because this character belongs to them both. Their appearance is of more conclusive import. If we can squeeze out small whitish plugs, consisting microscopically of squamous epithelium, the case is one of cancer. If the base consists of a uniformly reddish, varnished-looking tissue, which yields no plugs, it is probably a primary sore. The condition of the *lymphatic glands* is unimportant. Although the presence of hard enlarged glands is looked upon as confirmatory of the diagnosis of cancer, their very *absence* in this case may lead us to the same diagnosis. For a primary sore which has existed for a few weeks is invariably accompanied by enlarged glands, whereas they may not appear in cancer of the lip for many months.

We see, therefore, that the diagnosis of cancer of the lip, in the ulcerative stage, is easy. It is quite different, however, in the early stage, when the disease is either overlooked or neglected by the patient—a circumstance pardonable in the patient, but unpardonable in the medical attendant. If a person complains of a somewhat hard sore place, covered by a crust, on his lower lip, which bleeds on removal of the crust, we must not follow the ancient practice of touching the spot with caustic, instead of making a diagnosis. A spot which has been forming crusts for months, and which bleeds slightly, is not something which "may become malignant," as is often said as

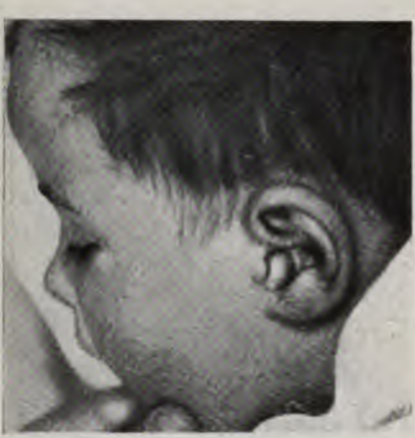


FIG. 34.—Dermoid of auditory meatus.

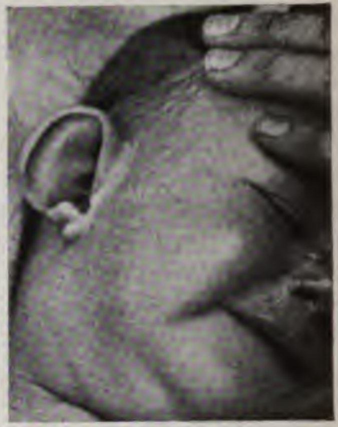


FIG. 35.—Deformity of external ear.

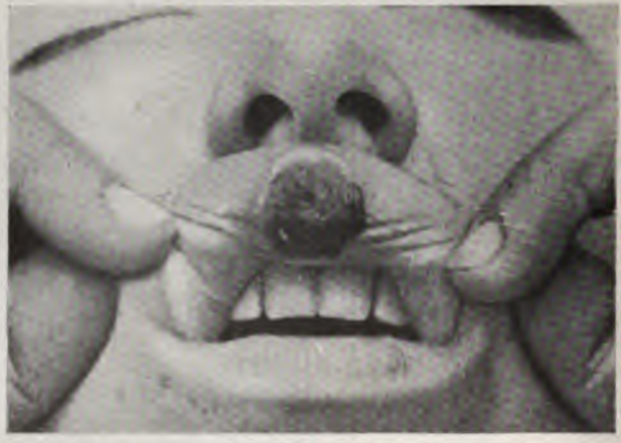


FIG. 36.—Primary sore of upper lip.

a matter of precaution, but is *actually* carcinoma, and must be radically removed without regard to the subsequent appearance of the lip, beard, or moustache. We usually see carcinoma of lip affecting the lower lip in males only (fig. 37), but occasionally the upper lip is attacked (fig. 38), and also the female sex, whether or not the habit of smoking a pipe has been indulged in.

(2) The *nose* is another favourite situation for ulcers. If the ulcer is in the vicinity of the nostrils, **lupus and tertiary syphilis** must be thought of first. An accurate history is extremely important in these cases, not only from a general clinical aspect, but also in regard to the development of the nasal disease. The most important rule is, that the various tuberculides of the skin, grouped together under the name of *lupus*, persist for months and even years, whereas the duration of *tertiary syphilides* is merely weeks, or at most a few months.

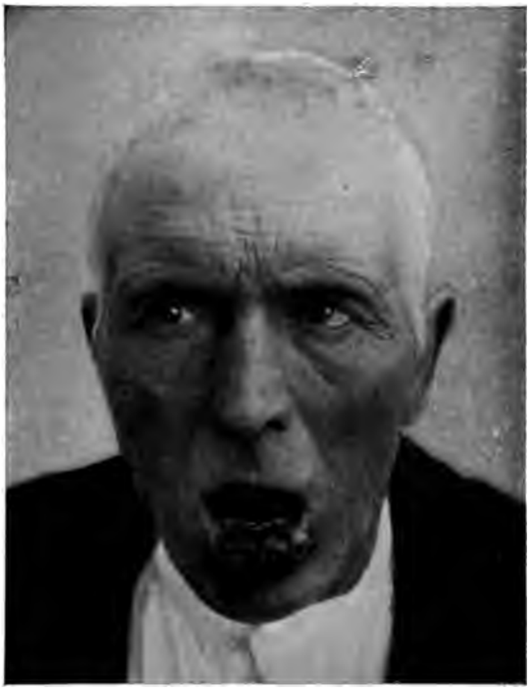


FIG. 37.—Cancer of lower lip.

In cases of lupus we may learn of occasional improvement, but never of spontaneous and complete cure. In cases of tertiary syphilis the patients will often—not always—tell of some exacerbations, with intervals of complete cure, marked, of course, by scar formation. If the patient is suffering from a first attack, the duration will have been much less than in a case of lupus, with a lesion of the same extent.

Let us now investigate the nose and its vicinity a little more closely. If, despite a long duration, the whole process is not accompanied by ulceration, we should at once think of lupus. But, on the other hand, the presence of ulceration is not conclusive, because both lupus and gumma can ulcerate. If the ulcer is serpiginous in outline, and has sharply defined edges with a fatty base, we must regard it as syphilitic. If, however, we find slightly bleeding ulcerated areas situated beneath scabs, with some grey nodules on these areas, we think of lupus. The recognition of these points, however, requires much practise. It is easy to draw conclusions from the *area surrounding* the infiltrated or ulcerated site, and, therefore, this requires very careful examination. If the change consists of separate very red nodules, nearer to peas than to lentils in size, which suppurate in the centre, or rather, become necrotic, running together into an irregularly circular ulcer, the case is syphilitic. But if we observe in the vicinity of a superficially eroded, brownish-red, soft, infiltrated area, separate little nodules hardly as big as lentils, situated under a normal or very scaly epidermis, and which change when pressed upon by a glass side into little light-brown spots, the nodules are certainly those of lupus. Their softness is very significant; a fine probe can easily be thrust within them from the epidermis without any violence. The *hypertrophic forms of lupus* must not be overlooked. These are characterized, besides the small nodules just described, by larger, soft, non-ulcerated nodules, or by thick, light, brownish-red plaques of soft tissue, several centimetres in extent.

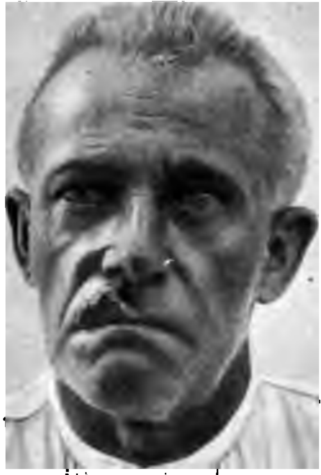


FIG. 38.—Cancer of upper lip.

We have not yet mentioned **cancer**. This disease does not generally attack the region of the nostrils, like lupus and syphilis, but rather the bridge of the nose, its lateral walls (figs. 39 and 40), and the naso-labial fold. The hardness of its margin and base distinguishes it immediately from lupus and syphilis, even in the chronic flat varieties.

As far as its *duration* is concerned, it may be said that an ulcer which has persisted for weeks, or for a few months, and which is constantly increasing in size, is always suspicious of cancer. But on the other hand, the fact that an ulcer has persisted for years, and has evinced signs of partial healing, or rather epithelization, is not always a conclusive argument *against* cancer. Indeed, we shall soon see that this course is significant of a definite variety of cancer. Although lupus frequently runs a similar course, the signs already given, and others which will be referred to subsequently, will, as a rule, prevent any confusion. The superficial cancer, known as *rodent ulcer*, often has a few little nodules on its smooth scars, just like lupus. But in contrast to the latter, they are whitish, hard, cannot be penetrated by the probe, and they do not leave the brownish stains on pressure with a glass slide. These little nodules appear to be isolated, but they are really arranged in a circular manner, and are mainly to be found at the edge of the cicatrized surface.

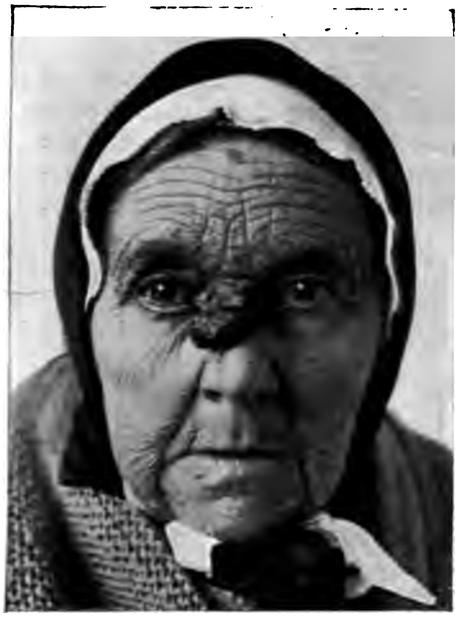


FIG. 39.—Tumour-like cancer of nose.

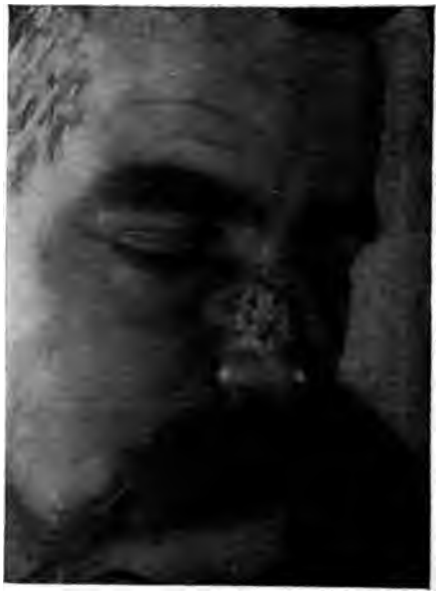


FIG. 40.—Cicatrizing flat cancer. (Rodent ulcer.)

Although many of these cancers are very benign, it is important to diagnose them before destruction of the deeper tissues occurs, and their *early stages* must be well recognized.

If we are consulted about a somewhat hard patch, scarcely raised above the surrounding part—a patch which is constantly scaling over, or is covered in the centre by a small crust, under which a slightly bleeding surface is evident, we can be confident that we are dealing with the form of skin cancer, designated as *rodent ulcer* (figs. 40 and 41). It is a condition which will gradually begin to invade the subjacent tissue, after lasting for years. Its oldest parts are characterized by central smooth scarring; its margin is of cartilaginous consistence, thick and whitish. There is always some contraction round about, causing the lips to be drawn up, the eyelids to be pulled down, or the cheeks to be drawn in, &c., according to the site of the new growth. The glands are rarely enlarged. If the disease has invaded the deeper structures, the skin becomes adherent to the underlying bone. Once this stage has developed the destruction continues slowly, but inevitably, involving the eyelids, invading the eyeball, eating into the nose and exposing its accessory sinuses, but nowhere causing any definite tumour formation.

Sometimes we are confronted with a semi-globular wart-like structure, which scales in the centre (fig. 42). Even in this condition the diagnosis is quite easy.

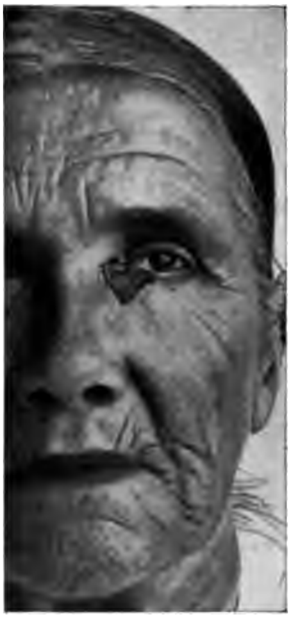


FIG. 41.—Cutaneous cancer of angle of eye. (Rodent ulcer.)

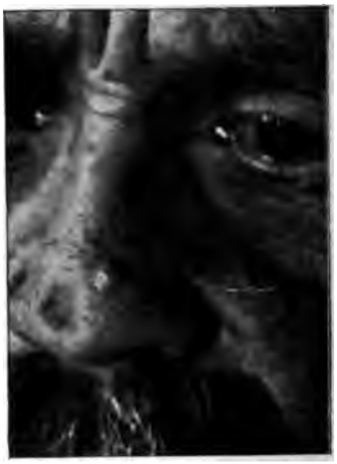


FIG. 42.—Wart-like cancroid of nose.

It is differentiated from a fibroma of skin, which is so frequent on the face, by its dense consistence and brief life-history. The absence of any inflammation and its sharply defined limits show that it is not of the nature of acne, while the scaling indicates some hyperactivity of the epithelium. If the wart has broken down into an ulcer with tumid proliferating edges (fig. 43), or if it has become a fungating or horny structure projecting from the skin (fig. 39), the diagnosis will be obvious, even to the inexperienced. The progress of this form

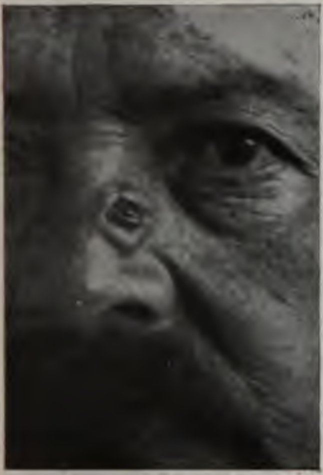


FIG. 43.—Cancroid of nose with wall-like margin.

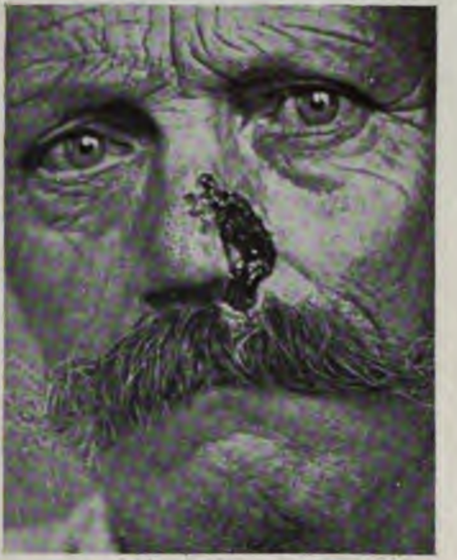


FIG. 44.—Rodent ulcer (basal cell cancer).

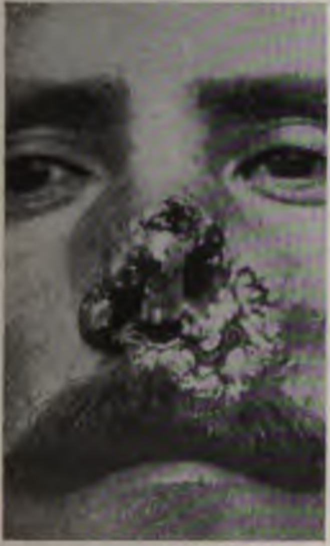


FIG. 45.—Cancer of nose on a lupus base.

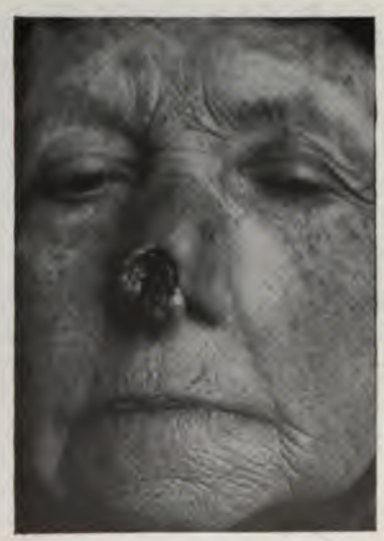


FIG. 46.—Lupus of the nose.

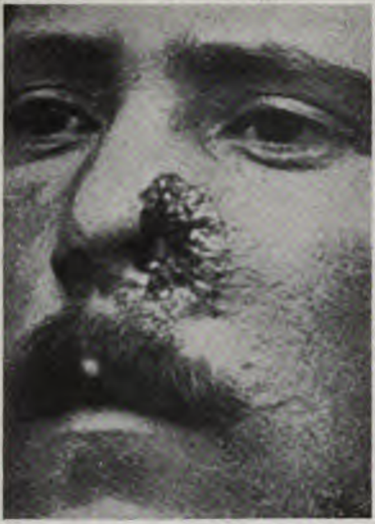


FIG. 47.—Tertiary syphilis of nose (resembling lupus).

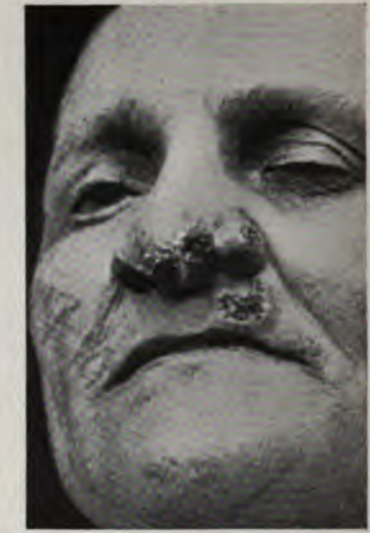


FIG. 48.—Tertiary syphilis of nose and lip.

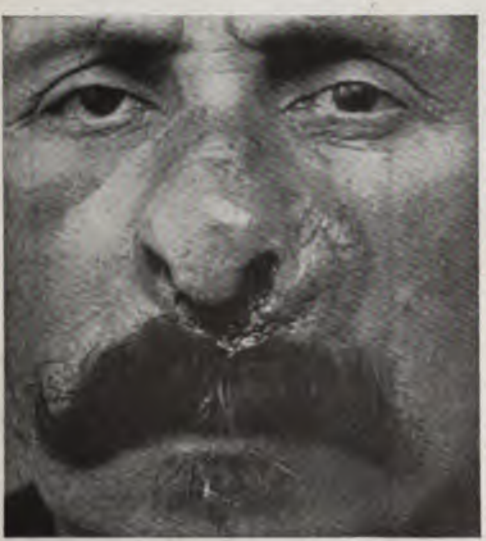


FIG. 49.—Sarcoma of nose.

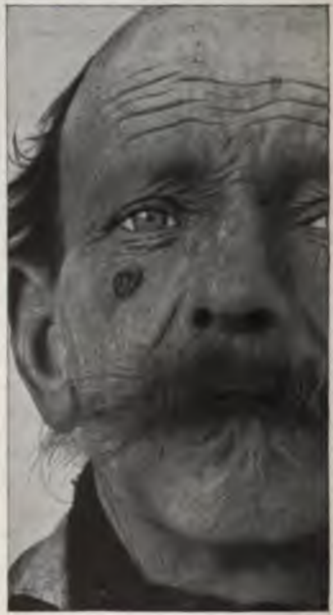


FIG. 50.—Rodent ulcer of cheek in early stage.

of cancer is quite different from that of rodent ulcer, because the destruction wrought by it in the course of *months* requires *years* in the case of the rodent ulcer. Histologically, this form of cancer originates in the horny layer of the skin and exhibits cancer "pearl-masses.' Further, the destruction is accompanied by much tumour formation, whereas this is not characteristic of rodent ulcer.

If the destructive process is far advanced on the nose, and the typical edges are absent, diagnosis without a histological examination is very difficult. Figs. 44 to 49 illustrate various conditions which may bring about the loss of the nasal appendage.

(3) We now come to ulcers, or structures which ulcerate early, in the **neighbourhood of the lids.**

A roundish little growth, varying in size from a hempseed to a pea, and somewhat drawn in at the top, must be looked on as very *suspicious of cancer.* It is distinguished from **molluscum contagiosum,** because the latter is softer and is rarely single, others being usually found close by, or towards the neck. If the contents are

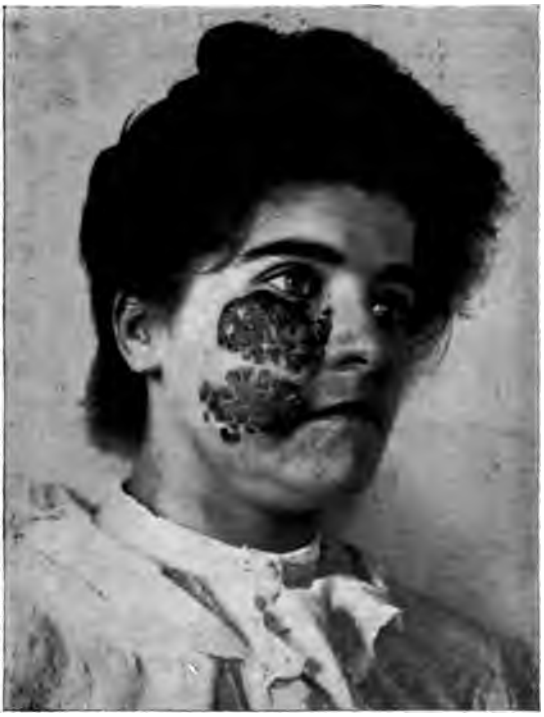


FIG. 51.—Lupus of the face.

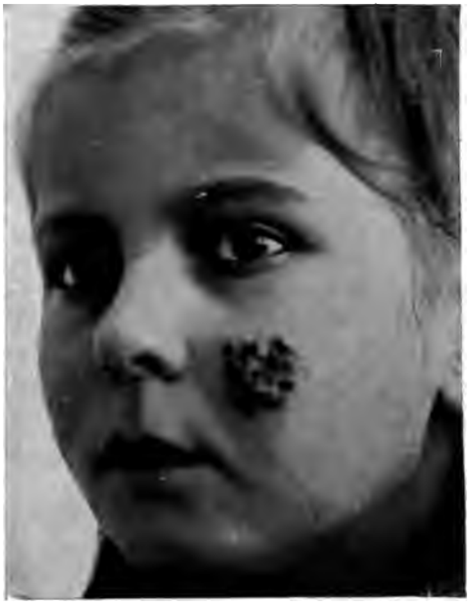


FIG. 52.—Lupus of the face.

expressed, they will be seen to resemble the interior of a sebaceous cyst, and under the microscope will show the well-known homogeneous oval molluscum bodies. If we squeeze an incipient cancer, we may obtain a few epithelial plugs, but we cannot empty the little tumour as we can a molluscum.

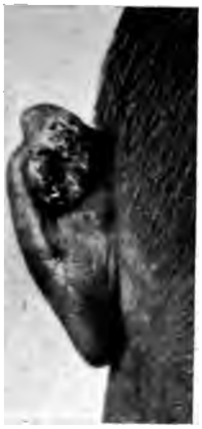


FIG. 53.—Cancroid of the ear.

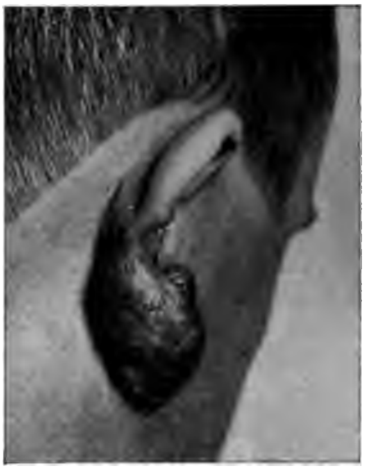


FIG. 54.—Tuberculosis of lobe of ear (Lupus hypertrophicus). (From the Ear Clinic, Basle.)

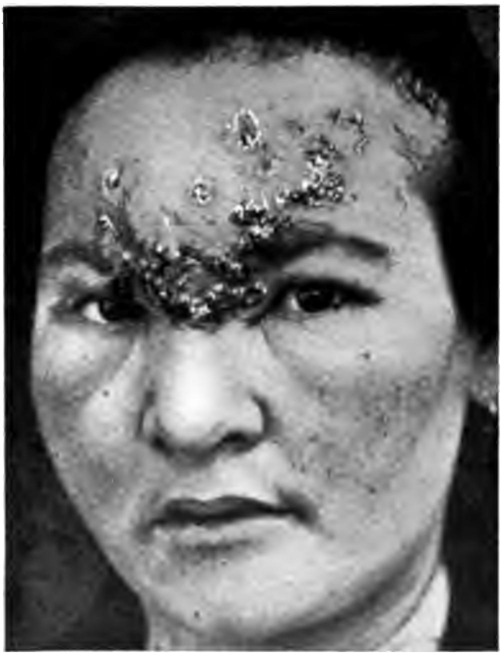


FIG. 55.—Tertiary syphilide of forehead.

(4) The remarks concerning growths of the nose and eyelids apply equally to the neighbourhood of the cheeks. Lupus is the most frequent lesion (figs. 51, 52,) and then comes *cancer*, mostly in the form of rodent ulcer (fig. 50). If all the facts are not consistent with a diagnosis of cancer, one must also think of molluscum contagiosum, just mentioned. In this connection the so-called telangiectatic granuloma should be referred to.

(5) Ulcers frequently form on the forehead and temple at the **margin of the hair.** These are usually **seborrhœic cancers of the skin.** They occur, as a rule, in old syphilitics, whose skins show very definite signs of seborrhœa and present other changes predisposing to cancerous induration, such as senile warts and even cutaneous horns. Such cancers may be multiple.

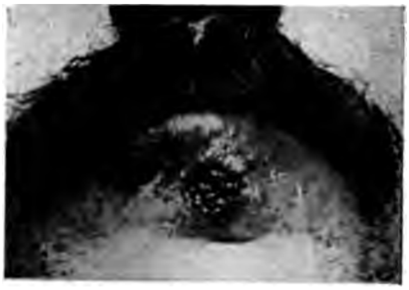


FIG. 56.—Primary chancre of lower lip.

In young persons an ulcer at the margin of the hair is very suggestive of an **ulcerated syphilide** (fig. 55).

(6) Ulcers of the **auricle** are generally cancroids. If left alone, they lead to complete destruction of the auricle (fig. 53).

Lupus in this position is more rare. In its hypertrophic, non-ulcerating form, it looks very much like a tumour (fig. 54).

Ulcers which have persisted for some time, not situated on the typical positions already referred to, may be suspected of another origin. For instance, if a patient, who is accustomed to patronize a barber, has recently acquired an ulcer on the chin or cheek, a **primary chancre** must be thought of, at any rate, so long as the asepsis of the barber has not reached the desired standard (fig. 56).

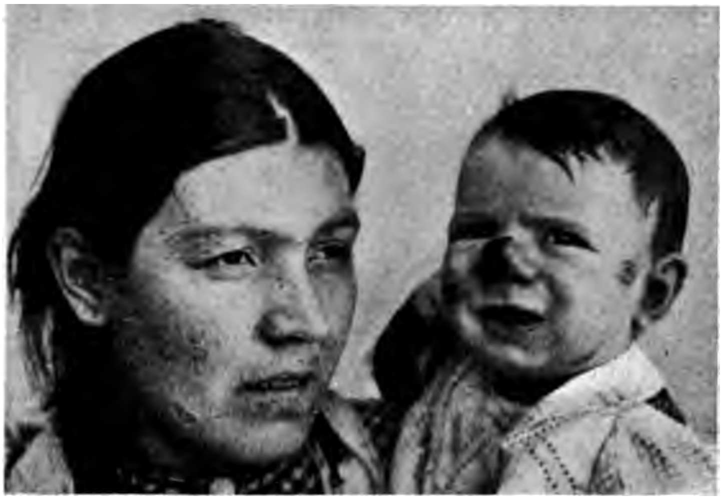


FIG. 57.—Oriental boil in various stages in child, and as a scar on the mother's cheek.

A primary chancre on the conjunctiva may lead to mistakes. A workman allowed his companion to remove a foreign body from his conjunctiva. A little piece of wood had been used for this purpose, which, according to a very dirty custom (to put it mildly) had been previously sucked by several people. The foreign body was successfully removed, but at the same time the spirochæte was inoculated.

There is a condition of great importance in the Tropics, where it is of frequent occurrence, known as oriental boil, Aleppo boil or Bagdad boil, etc. It is a skin infection produced by the protozoon of Leishmann. Fig. 57 illustrates the disease affecting a child, in its various stages, and the mother as a smooth scar on the right cheek.

CHAPTER XII.

INJURIES OF THE JAW.

FRACTURES of the lower jaw are so easily recognized that they hardly require any discussion. The most important question to ask is, whether the jaw has been entirely fractured transversely, or whether the alveolar process merely has been broken off. The severe disturbance of function, the position of the teeth, and lateral pressure and counter-pressure in the neighbourhood of both angles of the jaw, will answer this question without any difficulty.

A complete fracture may be overlooked when it is situated near the angle of the jaw or on the ascending ramus, that is to say, when it is outside the region of the teeth. But careful examination ought, as a rule, to detect it, because the loss of function and pain on local pressure are both very definite, even when there is no crepitus. The following case, however, gives an example to the contrary:—

A dentist, in extracting a wisdom tooth, unconsciously broke the jaw, which, in that situation, was somewhat atrophic. No conclusion could be drawn from the position of the teeth, because the fracture was at the angle of the jaw, and because there was no displacement. The patient only complained of severe neuralgia in the mandibular nerve, and this diverted attention from the injury to the bone, no suspicion being entertained that the nerve might be nipped between the two fragments. It was not until a phlegmon started at the seat of fracture that attention was directed to the accident.

A skiagram is necessary for the purpose of ascertaining the course of the fracture or fractures, with accuracy.

Fractures of the upper jaw more frequently escape recognition. Not, of course, fractures of the alveolar process due to violent extraction of teeth, or when the jaw is shattered by such an injury as a revolver shot for suicidal purposes. When all the bones of the face are torn asunder, the diagnosis of a fractured upper jaw presents neither difficulty nor interest. But, on the other hand, it is important to recognize a fissured fracture when the soft parts are uninjured and the seat of fracture not exposed. This may occur when a force is applied flat to the face, both upper and lower jaw being fractured transversely. The most striking symptom which comes on after the blow is bleeding from the mouth and nose; but this is not significant of fracture, because this may follow a simple wound of the nose. The presence of a rent in the mucous membrane, or at least a submucous hæmorrhage of the alveolar process of the palate, is more important. Preternatural mobility, or displacement of one of the fragments, would be conclusive evidence. But this is likely to be missed in a superficial examination, because the displacement is much less than in the case of the lower jaw. If, however, the patient complains that some of his teeth have "become too long" we may assume that we have overlooked some abnormality in position, and that a fracture is present. This very significant statement permits of the diagnosis being made long after the accident. Pain is also an important sign. It is easy to demonstrate by external pressure that there is pain on the anterior surface of the upper jaw, unless a hæmorrhage on the cheek prevents palpation. If such pain is limited to a definite position, we have every right to suppose that there is a fractnre. Sometimes the pain can be traced straight across to the opposite jaw. This supposition will be confirmed if pressure on the teeth either in an upward or lateral direction elicits definite pain, whether accompanied by crepitus or not. If there be neuralgia in the region of the infra-orbital nerve,

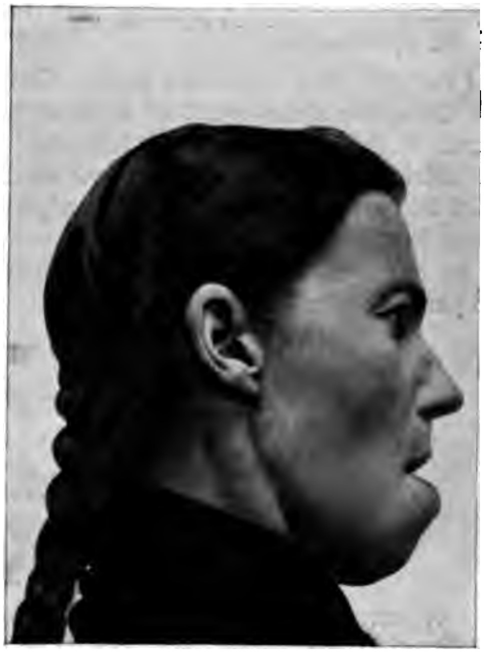


FIG. 58.—Dislocation of lower jaw. (From the Surgical Clinic at Berne.)

or possibly if sensibility be diminished, this affords further confirmation of the diagnosis.

The recognition of such fractures of the upper jaw, or even of less marked ones, has something more than a mere diagnostic interest, because the line of fracture may continue onwards to the base of the skull and involve other nerves, particularly the optic nerve. Such fractures may even lead to meningitis.

A woman was struck on the cheek by a drunken peasant. A few weeks later there still remained pain on pressure over the canine fossa, the feeling of long teeth, and a trigeminal neuralgia. Careful examination showed that the affected eye was blind, and the ophthalmoscope revealed optic atrophy, proving that the fracture had reached the area of the optic nerve.

Partsch has even seen a case of bilateral blindness from this cause.

Complete detachment of both upper jaws from the skull is very rare. I have however seen it follow a blow upon the face.

Dislocation of the lower jaw need not detain us. The patient with his lower jaw pushed forward, with his mouth open, unable to open it any wider or close it, presents such a characteristic aspect that even the layman can diagnose it forthwith (fig. 58). It is also easy to perceive whether the injury is unilateral or bilateral.

CHAPTER XIII.

LOCKJAW.

WHEREAS the inability to *shut* the mouth invariably points to dislocation, inability to *open* it may be due to various causes.

Let us begin with a rare case:—

A young girl was attacked suddenly, from time to time, with inexplicable seizures of lockjaw, which disappeared when the patient was semi-anæsthetized. This was evidently a functional condition, a spasm, which Kocher compares to spastic torticollis, and which we must assign to the wide domain of neuroses.

The rare cases of trismus, due to an apoplectic lesion in the lower frontal convolution, must also be classified as **lockjaw of nerve origin.** The lockjaw, due to tumours of the pons, is a peripheral irritative symptom.

The following kind of case is much more frequent: The patient had taken a three hours' walk from the country into town to consult a doctor, because he had been unable to open his mouth for the last fortnight, owing to "a bad tooth." Besides his lockjaw, he had facial paralysis of the left side, and a small unirritating scar over his left eyebrow. The latter was due to an injury from an axe, sustained four weeks previously. All the reflexes were increased, the gait was somewhat stiff, and there was a grinning facial expression on the unparalysed side (fig. 59).

This really constitutes the classical picture of **head tetanus**. The beginner, who thinks of the victim of tetanus as absolutely incapacitated, would probably be misled by the history of the three hours' walk. But patients with tetanus often come, walking to the doctor, from a distance. Facial paralysis is not always present, but in its absence the "risus sardonicus" is all the more expressive of the diagnosis, long before the patient has complained of his malady, especially of his bitten teeth. (Fig. 61).

If these somewhat infrequent causes do not account for the lock-jaw, it is necessary to determine some anatomical causation.

We will proceed with the examination in the natural method. If the patient's cheek has recently become swollen, and if his expression is typical of toothache, we may suspect **periostitis of the jaw.** The mouth is opened as widely as possible, and the offending tooth, probably one of the molars, is looked for. There may be nothing wrong to be detected with the tooth, but the fold of mucous membrane between the jaw and the cheek will be more or less obliterated.

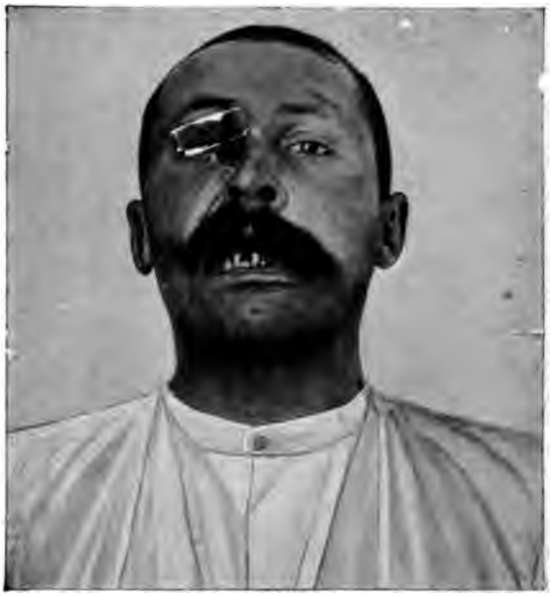


FIG. 59.—Head tetanus. The patient is attempting to open his mouth. Contraction of right side; facial palsy on the left. Scar over left lid.

Instead of an even and regular swelling of the cheek, there may be a dense infiltration, with sundry sinuses, foci of granulation and fibrous contractures, reaching down as far as the neck (fig. 63). This will at once suggest **actinomycosis.** The pus expressed from the sinuses frequently contains the well-known yellowish little granules, of the size of millet seeds, which serve to confirm the clinical diagnosis.

We shall see later on that Actinomycosis or its allied Streptomycosis may also begin as a diffuse swelling.

Tuberculosis of the jaw, which somewhat resembles the above, occurs but rarely. The first glance should, however, differentiate it, because the skin is less involved and because the glands are considerably enlarged.

If the lockjaw has set in acutely, and a diffuse swelling occupies the whole of the lower jaw and the floor of the mouth, we should think of **osteomyelitis of the jaw.**

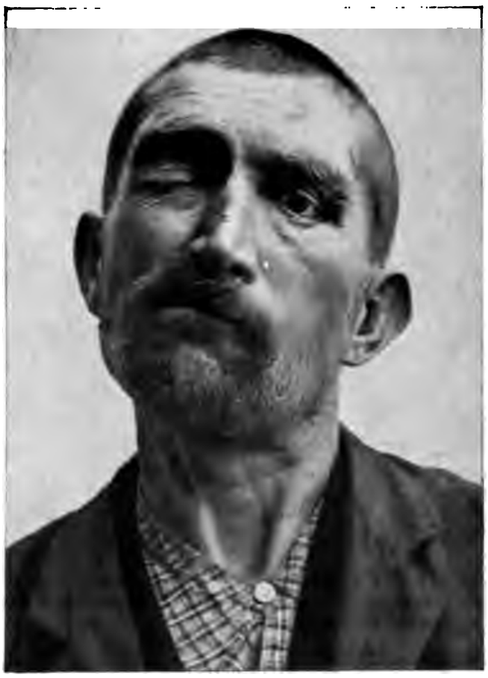


FIG. 60.—Head tetanus, with facial palsy. Small wound on the left eyebrow.

A swelling more closely confined to the cheeks, with tumid lips and a fœtid, slightly sanious fluid issuing from the mouth, points to one of those rare cases of **gangrenous stomatitis.** *Mercurial stomatitis* may produce a similar clinical picture.

If the aspect of the patient does not suggest any cause for the lock-jaw, we must examine the condition of the **articulation of the jaw.** A rapid and painful onset of the trismus will make us think of *acute arthritis,* which occurs in the most varied infective diseases, especially in scarlet fever, acute articular rheumatism and gonorrhœa. The region of the joint appears somewhat swollen, pressure in front of the ear, just under the zygomatic arch, is painful, and the patient complains of radiating pains in the neighbourhood of the joint. Every attempt to open the jaws forcibly produces immediate contraction of the muscles of mastication. If there are no acute inflammatory signs and the lockjaw is of old standing, but has followed some acute disease, we must conclude that the articulation of the jaw has become ankylosed through some *antecedent inflammation.*

It must not be forgotten that ankylosis of the jaw can develop in the course of a chronic ankylosing polyarthritis.

A very trifling derangement in this region may be mentioned here. It consists of some crepitation in the joint due to a slight looseness of the capsule of the articular condyle (termed discitis by Lanz). In other cases this crepitation points to arthritis deformans of the jaw, which proceeds, if it is unilateral, to cause obliquity of the part, with corresponding obliquity of the lower teeth.

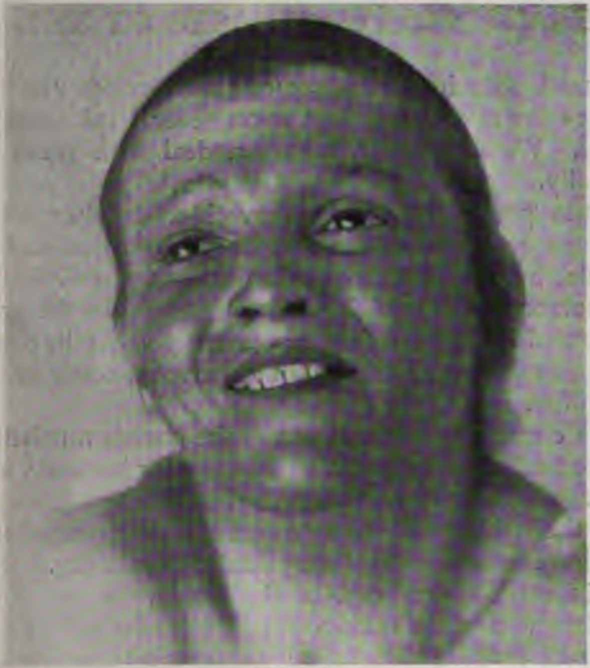


FIG. 61.—Risus sardonicus of tetanus.

Among the causes of ankylosis of the jaw are **inflammatory processes in the neighbourhood,** which attack the jaw secondarily. This occurs especially in *suppuration of the tympanic cavity*. **Injuries** also play their part, but they may lead to bony proliferation or to the development of osteomata.

A striking smallness of the lower jaw—a bird face—accompanying the lock-jaw, will indicate that the disease dates from infancy. Disuse of bone leads to atrophy, and therefore the ankylosed lower jaw fails to grow adequately.

If the exhaustive examination of the joint and its surroundings fails to discover the cause, we must inspect the pharynx. If the disease is recent, it may be due to a *retro-tonsillar abscess;* if chronic, it may be due to *cicatricial bands* in or under the mucous membrane, or to a *malignant neoplasm* near the tonsil.

CHAPTER XIV.

INFLAMMATORY DISEASES OF THE JAW.

In discussing the important symptoms of lock-jaw in the preceding chapter, we have already touched upon several inflammatory diseases of the jaw. We must now, however, deal with them again, more systematically.

(1) **Acute periostitis** of the jaw, which is always the result of dental disease, presents such a familiar picture that its consideration need not detain us. It can only be necessary to distinguish it from **primary osteomyelitis**, which will be suggested by the great extent of the disease and inability to find any primary lesion in the teeth. The course of this disease resembles, in its severity, acute osteomyelitis of the extremities.

(2) The **chronic inflammations** of the jaw are much more interesting. If we find a limited area of disease, for instance, a sinus in the gum or on the cheek, with more or less swelling on the corresponding section of the jaw, we may be sure that a tooth is responsible and that the condition is one of an ordinary **dental fistula** (fig. 62). The more contracted the neighbourhood of the fistula, the longer will its duration have been. If there is no tooth in the affected situation, a piece of stump will probably be found on opening the alveolus, or in an extensive case a *sequestrum* may be forthcoming. If the removal of such causes does not cure the inflammation, some more serious disease underlies it, and we shall have to diagnose between *actinomycosis, tubercle,* and *phosphorous necrosis.*

The symptoms of *actinomycosis of the jaw* have been briefly described in the previous chapter; it only remains to add that the changes at the site of infection—a tooth or the gum—are so completely overshadowed by the secondary appearances on the cheek and neck, that it frequently becomes impossible to define the spot where the infection entered. But it is interesting to trace *the source of infection.* Usually we can find no clue in the case of the town dweller, and in country districts the bad habit of chewing stalks of grass is so widespread that the patient is most unlikely to remember any such incident. The clue is more definite, if one can ascertain that the patient has been tending cattle with actinomycosis, as once occurred to me in a case of abdominal actinomycosis.

The diagnosis is based on the visible external changes (figs. 63 and 64), on the dense infiltration with brownish-red foci of softening on the somewhat contracted cicatricial areas, on the absence of glandular enlargement, and finally on the demonstration of the little yellowish granules.

The latter may be confused with the small particles of necrotic tissue which sometimes occur in tubercular abscesses. They may, however, be pretty clearly distinguished, even without the microscope, in the following manner: If a granule of the ray fungus is crushed between two glass slides, and examined, the periphery will be seen to be opaque from pus cells, while the centre will be comparatively transparent, being occupied by the interlacing fibres of the fungus. On the other hand, a pus coagulum, or a particle of necrotic tissue, will be uniformly turbid.

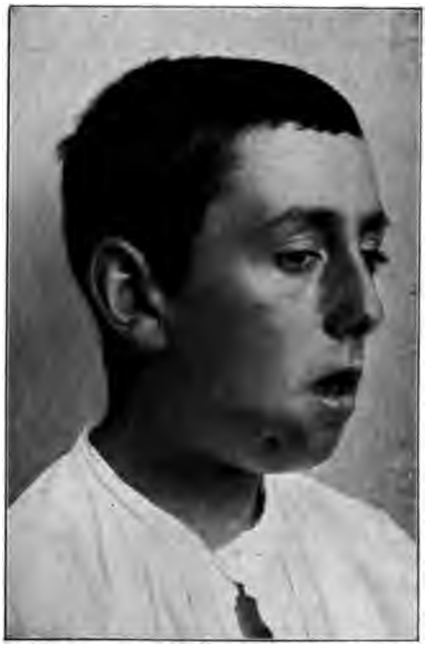


Fig. 62.—Sinus from tooth.

When all the described signs of actinomycosis are fully developed, a diagnosis can be made at first sight, even if the characteristic granules are not found. But, on the other hand, actinomycosis must not be excluded if there is only a purely diffuse swelling present, without the skin changes (fig. 63). The pus should be examined for the ray fungus (actinomycosis or streptomycosis) in the case of every persistent chronic abscess of the jaw with hard edges. If the immediate examination yields a doubtful result, culture tests should be undertaken.

We have already referred to **tuberculosis of the jaw** among the causes of lockjaw. It may appear in the form of *tubercular ulcers of the gum.* These are diagnosed by their sharp margins, chronic course, the presence of enlarged soft glands in the neck, and the failure of antisyphilitic remedies. To expedite the diagnosis, it is necessary to scrape away a piece of the margin of the ulcer with a sharp spoon, and it will generally be easy to detect tubercle in the frozen sections. In doubtful cases animal inoculations will yield a decisive result.

The early stages of *tubercular disease of the bone itself* are more difficult to recognize.

The following is a characteristic case :—

A woman, aged 38, gradually became affected with lock-jaw. Already at the first examination severe swelling of the cheek and temple were present. Some of the molars were absent, and the others did not appear to account for the clinical condition, more especially as the bone was thickened, especially about the angle of the jaw, and the ascending ramus. A little pus exuded from a small sinus behind the last molar, and the probe impinged upon bare bone. A bunch of movable soft and elastic enlarged glands was found in the neck. The swelling over the temple fluctuated.

Four points in this case established the diagnosis forthwith :—

(1) The insidious onset, which distinguished the disease from an ordinary dental periostitis.

(2) The localization on the ascending ramus, which is not usual in dental periostitis.

(3) The obviously tubercular glands in the neck.

(4) The presence of a cold abscess over the temple. This abscess, which would be called a gravitation abscess if it were not making its way upwards, is quite distinctive of tubercle of the lower jaw. Owing to anatomical reasons the pus cannot find its way downwards; it tracks along the line of least resistance, between the pterygoids and the bone, makes its way upwards and gets beneath the temporal muscle. The temporal bone and other bones of the skull may be attacked secondarily as a result of this abscess.

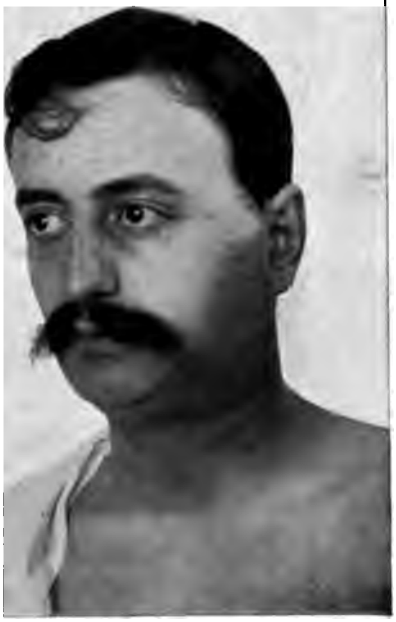


FIG. 63.—Early stage of actinomycosis of jaw (before the bursting of abscess).

The subsequent progress of the above case was also very distinctive.

The abscess was opened from the mouth, a tubercular sequestrum was removed from the ascending ramus and the bony cavities were scraped out. But this only gave temporary relief. Before the disease was checked it was necessary to resect the whole of the ascending ramus, and subsequently a portion of the horizontal ramus. The tubercle afterwards invaded the upper jaw, and this had to be resected; and two years later the patient had ileo-sacral tuberculosis of both sides.

Most cases of tubercle of the jaw, hitherto described, have run a similar course.

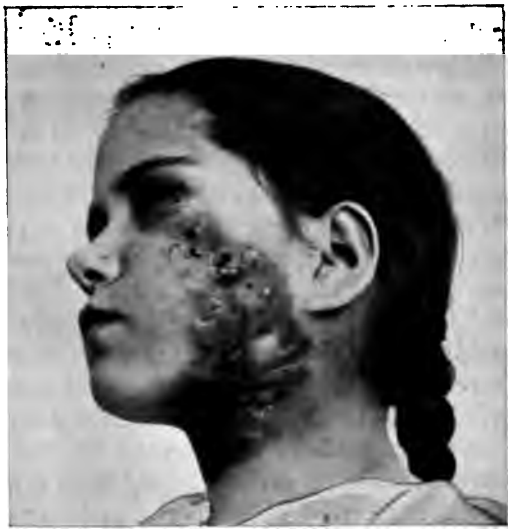


FIG. 64.—Actinomycosis of jaw. (Advanced stage.)

Phosphorus necrosis presents a different picture altogether; the trouble also begins with insignificant symptoms of toothache, but the extraction of the painful teeth does no good. The pain persists, one acute abscess after another appears and they burst, either through the gum or externally, leaving permanent fistulæ. The whole jaw becomes diffusely thickened through the formation of new bone from the periosteum, and finally a piece of the original jaw extrudes itself — the whole lower jaw may indeed become necrosed—and is easily removed by the patient himself or the medical attendant.

In both phosphorus necrosis and osteomyelitis of the jaw extensive sequestra may form; but in phosphorus necrosis this process does not develop from one sudden attack, but is the result of slow stages. Tubercle is similar to phosphorus necrosis in its chronicity, but the latter differs completely in the large extent of sequestrum formation. The only similarity between phosphorus necrosis and actinomycosis is also their chronicity. But the former always remains a bone disease despite abscesses in the soft tissues and despite fistulæ; in actinomycosis the affection of the bone assumes minor importance and the disease of the soft parts predominates. It is obvious that it must be shown that the patient has had long contact with phosphorus in order to confirm the diagnosis of phosphorus necrosis. But it must not be forgotten that the disease may arise many years after the patient has ceased to be under the influence of phosphorus. Phosphorus necrosis might happily become merely a matter of history, if new spheres for the employment of yellow phosphorus in industry were not being opened up.

CHAPTER XV.

TUMOURS OF THE UPPER JAW.

Tumours of the upper jaw are imitated by harmless conditions, just as are so many malignant diseases, a circumstance responsible for delayed diagnosis. *Periostitis of the jaw* and *chronic inflammation of the maxillary sinus* are the principal ones. A swelling in the upper jaw, coming on with toothache, sends the patient at first to the dentist, and a few teeth are extracted. If he is disturbed by unusual nasal discharge or by the obstruction of a nostril, he consults a nose specialist to get his antrum washed out. The patient is fortunate if it is recognized that his case does not belong to those specialities, but concerns the surgeon. This summary diagnosis is not difficult if one appreciates the maxim that *any swelling of the upper jaw, however small, accompanied by persistent neuralgic pains, must be suspected of a malignancy.* Other symptoms may be entirely absent, such as displacement of the nose, a sanio-purulent discharge from the antrum, deviation of the eye (squint, double vision), ulceration into the oral cavity or vestibule of the mouth. Nevertheless, there can be no doubt of the diagnosis, if on comparing both sides it is found that the canine fossa is somewhat obliterated, the lower orbital margin spherical or irregular, the floor of the orbit perhaps already raised, and if the neuralgia is of infra-orbital character ; especially if one of the other symptoms just mentioned is also present. To confirm the diagnosis, some of the diseased tissue should be removed with a sharp spoon from the antrum, either through the nose, or by making a small incision and opening the antrum from the vestibule of the mouth. The piece removed must be submitted to histological examination, which will at the same time reveal the intimate structure of the growth. Clinical diagnosis cannot go beyond declaring the presence of a *malignant growth*, it cannot define its histological details. Theoretically we should expect that a tumour which at first is confined within the antrum and then gradually breaks through its wall is more likely to be a periosteal sarcoma—a growth which rapidly makes its way externally—than a squamous epithelioma. Experience, however, shows that the very opposite may occur. Sometimes sarcomata are limited to the antrum for months, and squamous epitheliomata may proliferate so rapidly towards the surface that warm poultices are ordered on the assumption that the swelling is due to periostitis. Palpation affords no conclusive evidence. In the case of tumours of the upper jaw it is not possible to rely on the distinction that firm nodular growths are generally cancerous, and that round, elastic growths are usually sarcomatous. Early enlargement of the glands is more important for the diagnosis of cancer, but this sign is often absent.

But the practitioner is under no obligation to worry himself over the differential diagnosis. He has done his duty if he has sent the case, in an early stage, to the surgeon as one of malignant growth. From the operative point of view it is immaterial whether it be sarcoma or carcinoma. There is, however, a great difference in the prognosis, as the ultimate results of operations on sarcomata of the upper jaw are much better than those on carcinomata.

As we have already mentioned, difficulties of diagnosis may arise from *empyema of the antrum*, *periostitis of the jaw*, *tuberculosis of the jaw*, and *dental cysts*. Each one of these diseases has its characteristic previous history.

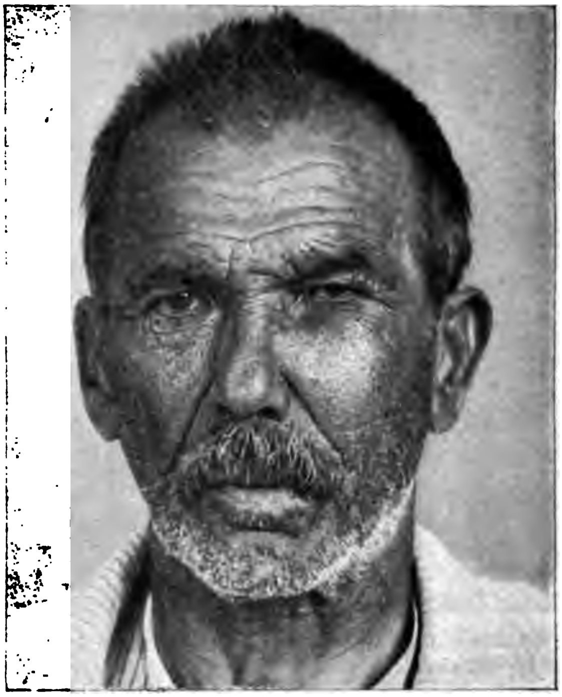


FIG. 65.—Carcinoma of upper jaw.

In **chronic empyema,** the most striking incident is the periodical discharge of pus. As long as the discharge is free, there is absolutely no pain, but when it is retained there is a neuralgia of racking, beating, and sometimes of a boring character. There is also local pain on pressure in the stage of retention. During the intermediate stage the pain is dull and quite tolerable. But in the case of malignant growths, the pain is severe and tormenting, and allows of no respite, even when there is no external sign of irritation or any marked pain on pressure. There is sometimes a purulent discharge in carcinoma, and this may lead to the mistaken diagnosis of ordinary antrum disease. But the fact that there is so much pain in the presence of a free discharge must arouse the suspicion of a malignant neoplasm.

It is usually easy to detect the offending tooth in a case of peri-

ostitis of the jaw. Malignant tumours produce toothache in teeth which are otherwise healthy. The pain also involves the cutaneous. branches of the nerves (upper lip) and is frequently accompanied by anæsthesia (anæsthesia dolorosa). In both periostitis of the jaw and in empyema of the jaw the pain is only present in the acute stage, or in acute exacerbations, and it becomes relieved either spontaneously or after appropriate treatment. One special form of ostitis can give rise to very serious difficulties in diagnosis, namely, **tuberculosis of the jaw.** Its characteristics have been mentioned above.

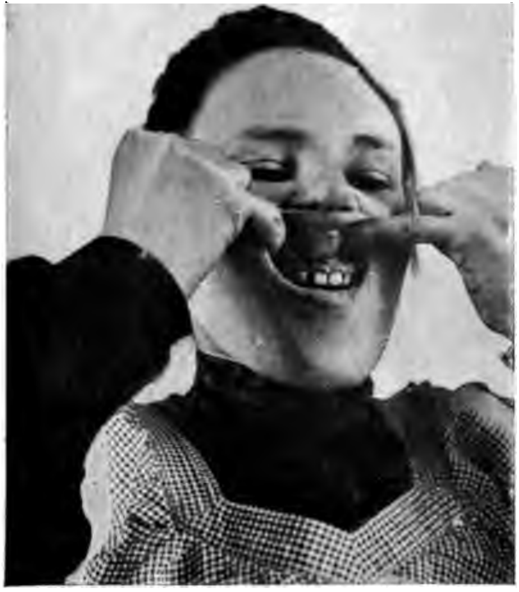


FIG. 66.—Dental cyst on upper jaw. The right lateral incisor is absent below the cyst.

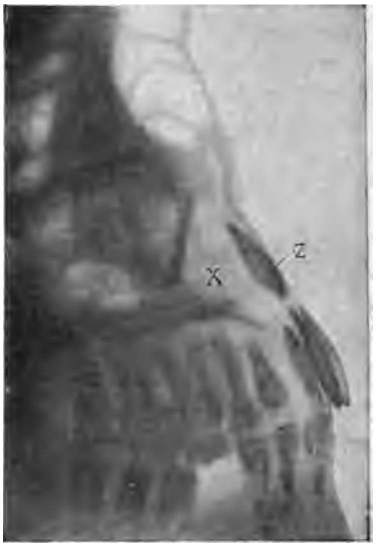


FIG. 67.—Skiagram of fig. 66. *X*, Dental cyst with the missing incisor tooth, *Z*.

In cases of **cysts of the jaw,** there is a gradual enlargement of the bone, which eventually crumbles like parchment, but there is usually no pain as long as the contents of the cysts are not infected. In some cases they empty themselves periodically through the nose and then fill up again. They may persist for years, a circumstance which differentiates them from malignant growths, for both in carcinoma and in sarcoma the diagnosis can generally be firmly established within a few months of the onset of symptoms. If one tooth is missing from the set and is seen, on the skiagram, in the tumour, all doubt is dispelled (*see* figs. 66 and 67).

If we meet with the symptoms of a malignant growth of the upper jaw in an adolescent male, we must also think of a **nasopharyngeal fibroma.** This growth springs from the base of the skull near the roof of the pharynx and penetrates into all accessible fissures, especially into the nose, the orbit, and circuitously into the parotid region and the maxillary sinus.

I have seen a case wherein the tumour penetrated thence into the mouth cavity and began to putrefy. This peculiar course at once suggested sarcoma. Histological examination and further progress showed, however, that it was an ordinary nasopharyngeal fibroma. The patient made a good recovery, although the tumour was not completely extirpated from the base of the skull, it only being possible to burn it away with the thermocautery.

Innocent tumours of the body of the upper jaw, apart from dental cysts, are so rare that they do not enter seriously into the question of differential diagnosis. They behave like the corresponding growths of the lower jaw, the description of which should be referred to.

On the other hand, *innocent tumours*, or *growths only malignant locally*, play the most important part in connection with the *alveolar process*. These will be discussed, together with the growths of the oral cavity.

CHAPTER XVI.

TUMOURS OF THE LOWER JAW.

APART from neoplasms of the gums and alveolar process, which we shall discuss in connection with tumours of the oral cavity, new growths of the lower jaw present no difficulties of recognition. Care must be taken, however, not to confuse a swelling which appears to be a *tumour* with what is really an *inflammatory process*. We must abandon the thought of a new growth, and confine ourselves to one of the inflammatory conditions described in Chapter XIV: if the disease has started with toothache caused by carious teeth which are in evidence; if the thickening of the jaw has been preceded by an acute inflammatory stage; if the patient tells of repeated acute exacerbations and shows the scar of an old dental fistula in testimony thereof; or if we find tubercular glands of the neck in a purely chronic disease. We are, however, justified in diagnosing forthwith a new growth: if the swelling has come on gradually and painlessly;

if toothache, when present, is a late symptom, and healthy teeth become loose without any visible inflammatory changes in the gum. Palpation will sometimes elucidate the condition. An inflammatory swelling gradually merges with the healthy bone, whereas most tumours appear to be sharply separated therefrom. Central tumours form an exception to this rule, because these, at first, expand the bone in a spindle-shaped manner. In these cases, the exclusion of any antecedent dental disorder is decisive from the point of view of diagnosis.

Having concluded that a growth is present, the first question concerns its innocence or malignancy. The history supplies the critical factor, because a *slow growth* always means innocence, and a *rapid* growth signifies malignancy. But this criterion must be cautiously applied, because even a sarcoma may last for many years. It is necessary, next, to consider the matter of *painfulness*. Painless tumours, which remain painless for years, are innocent. If toothache comes on, a suspicion of malignancy arises, but only suspicion, for innocent growths may cause neuralgia by pressure on the inferior dental nerve. On the other hand, victims of sarcoma may remain free from pain for a considerable time. It is only in extreme cases, therefore, that the previous history permits of the formation of an immediate diagnosis. In intermediate cases every clinical aid will need to be enlisted, and often enough the microscope will be required to make the final decision. It must not be forgotten that a tumour which has been innocent for years may eventually become malignant. This change can generally be recognized by sudden rapidity of growth.

We have purposely omitted one sign, viz., the absence or presence of metastases in the glands of the neck, because this is not a matter of significance in connection with tumours of the lower jaw. Malignant growths in this situation are *sarcomata*, which usually leave the glands unaffected. Enlarged glands should therefore not be regarded as a mark of malignancy, but, as previously stated, rather an indication of tubercular disease.

We now proceed to discuss the individual varieties :—

(1) The first group of growths of the lower jaw embraces tumours connected in some manner with the *development of the teeth*, or at any rate with the *epithelial covering of the jaw*. They are not indeed of frequent occurrence, but they are of great theoretical interest. Among these are dental cysts, which appear during the period of youth ; they push the jaw *outwards*, and they contain either a tooth which is missing from the series, or a supernumerary tooth. The expansion of the jaw which they cause is very gradual, so that finally the bone gives the crumbling sensation peculiar to parchment. If such a cyst

has been incised in ignorance of its true nature, or if it has burst spontaneously, a fistula remains which is very prone to secondary infection, wherewith the whole aspect of the case is obliterated. Another group consists of *odontoma* and *adamantinoma*. These may be soft, or as hard as enamel, or of a mixed character. They arise in young people as an irregular proliferation of the various elements of a tooth, and they gradually, but painlessly, displace the enclosing bone, both *outwards and inwards*. Their favourite site is in the neighbourhood of the posterior molars (fig. 68). Another tumour, less associated with tooth development, is **the multilocular cystoma of jaw,** in which the bone becomes gradually expanded by cystic proliferation of its epithelial covering, and finally becomes converted into a vesicular shapeless structure. This change eventually attacks the ascending ramus of the jaw, in contrast to what occurs in simple cysts of the jaw and odontomata.

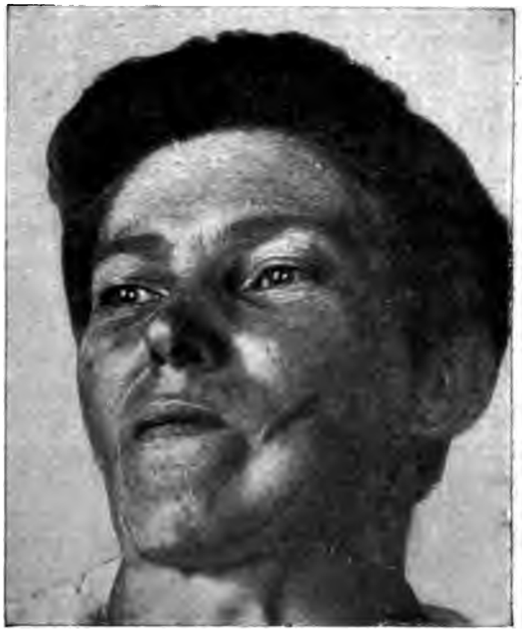


FIG. 68.—Odontoma of lower jaw.

(2) Turning now to *connective tissue tumours*, we must mention the *innocent growths*, **fibroma, chondroma** and **osteoma,** as of comparatively rare occurrence. If they arise from the surface of the bone, they grow slowly, their structure is nodular, they feel firm or hard, and only become troublesome through secondary changes. But if they exist within the bone, their pressure on the inferior dental nerve may soon produce neuralgia. The bone is expanded, first in a spindle-shaped manner, and having reached the surface the growth appears as a nodular sharply defined structure.

Sarcomata, *malignant connective tissue tumours*, play the chief *rôle* among the tumours of the lower jaw. The initial symptoms and early discomfort depend upon whether their site is central or peripheral; but their course is more rapid than that of the tumours previously discussed, even if, exceptionally, the first signs date some years back.

The following observation is very significant:—

A female, aged 50, came to her medical attendant complaining of a gradual thickening of the right horizontal ramus of the lower jaw. It was regarded as a dental cyst and was opened from the mouth. The knife penetrated into a hollow space, from which a profuse stream of blood flowed. I saw the patient, two years later, with a diffuse expansion of the whole right half of the jaw. The diagnosis pointed to sarcoma, and the operation consisted of removing half of the jaw, which was expanded as far as the articular process into vesicular cavities, just like a multilocular cystoma of the jaw. The small amount of firm tissue present showed the structure of a round-celled sarcoma. Two years subsequently I saw the patient again. Locally she was cured, but there were secondary deposits of similar structure in the skull and sternum.

The diagnosis may be difficult in the early stages, as already intimated, but it cannot be missed if the tumour has proliferated into the oral cavity, has caused the teeth to fall out, has become adherent to the skin, or has finally ulcerated to one side or another. It should, however, not be allowed to reach such a degree; every tumour of the jaw which does not remain quite stationary ought to be suspected and removed.

A careful examination will generally reveal the starting point of the tumour, notwithstanding many exceptions. The sensation of crinkling like parchment indicates a central site; the involvement of bone in the growth used to be detected by the acupuncture needle, but nowadays Röntgen-ray examination replaces this procedure, with greater accuracy.

We have said nothing about *cancer of the lower jaw*, but this will be referred to in the next chapter, as it usually grows from the mucous membrane of the gums. Isolated masses of dental epithelium, deeply *displaced*, very rarely undergo cancerous degeneration.

CHAPTER XVII.

ACUTE INFLAMMATORY DISEASES OF THE ORAL CAVITY.

We have already met with some of the acute inflammatory diseases of the interior of the mouth, and we shall come across them again when we discuss some of their main symptoms: lockjaw and difficulties in swallowing and breathing. We shall, therefore, here only collate a few points which have been treated disconnectedly, and supply sundry omissions.

An acute swelling of the *lips* nearly always depends upon the presence of a small **furuncle,** which may have disappeared in the general œdema, assuming, of course, that the swelling is not a part of an extensive inflammation like erysipelas, or the result of a periostitis of the jaw. We have already noted the possible dangers of a furuncle of the lip.

An *acute circumscribed swelling of the gum* indicates either a diseased tooth or a root retained in the alveolus. The precise localization is shown by the position of the most intense redness of the mucous membrane, and the greatest obliteration of the fold between the cheek and jaw. If the offending tooth is still visible, it will readily respond to light percussion.

Acute widespread swelling of the gum is a sign of some form of acute periostitis of the jaw (*see* lower jaw), or of a general stomatitis (*see* below).

Acute swelling of the *floor of the mouth* may be due to many causes. If the swelling is in the middle line, and more definite posteriorly than anteriorly, we should think of a secondarily **suppurating dermoid,** which is common in children, or of an inflamed lymphangioma. In such a case an adult would observe that there had been something abnormal under the tongue before the onset of the acute inflammation.

If the inflammation has proceeded externally towards the submental region, the case is one of **phlegmonous submental lymphadenitis.** A sore on the lip, an acute pustule, a small infected abrasion of the skin, will have afforded the portal of entry of the infection.

If the swelling on the floor of the mouth is rather unilateral, or if the patient can state on which side it began, **acute inflammation of the salivary glands** must be thought of. If the swelling is anterior, the sublingual gland is affected; if lateral, it is the submaxillary gland. In these cases the floor of the mouth, in the vicinity of the gland, may look like a translucent œdema and the swelling feel like a board. The cause of the swelling will be found in a salivary calculus, especially if the patient states that he has suffered from repeated attacks of such inflammation. If these exacerbations are of short duration, and terminate with a discharge of saliva, there is no doubt the swelling indicates salivary retention. If they are of longer duration—a day or more—and increasing infiltration of the tissue takes place, we must conclude that some bacterial inflammation of the salivary glands has become engrafted on the simple retention. If treatment is not undertaken, the swelling will proceed to its natural result—the formation of an abscess.

If the symptoms are distinctly inflammatory from the start and are better developed externally than towards the mouth cavity, the case is one of **acute submaxillary lymphadenitis.** Its cause will most probably be found on the gum, or the cheek, or nose if the portal of entry still remain demonstrable.

If the infection is very severe, and tends to spread, especially towards the floor of the mouth, it is termed "Ludwig's angina," which is merely a clinical and not an etiological indication. The exciting causes, as usual about the mouth, are staphylococci, streptococci and colon bacilli. The great severity of the infection strongly suggests that the infection arises from some deep focus—directly around the submaxillary gland.

If the swelling involves the *tongue* from the start, and this organ quickly becomes converted into a dense immovable mass, with saliva trickling from the open mouth of the patient, who can neither swallow nor speak, but can just breathe, the case is one of **acute glossitis,** which usually ends with the formation of a lingual abscess. This rare disease is of a metastatic character, and supervenes especially as a sequela of acute infectious diseases. If in the course of glossitis there be dyspnœa with delayed respiration and stridor, it is obvious that the larynx has become œdematous, and that tracheotomy is urgently required. If, on the other hand, the dyspnœa is accompanied by cyanosis and hurried respiration, without stridor, we conclude that aspiration pneumonia has supervened, and our prognosis will be correspondingly doubtful, if not bad.

Sometimes the entire floor of the mouth is involved in a phlegmonous inflammation from the very beginning; and the tongue, which is usually œdematous at the same time, is pressed against the palate. The clinical picture resembles that of Ludwig's angina, but is bilateral from the start. This constitutes a case of **acute phlegmon of the floor of the mouth,** and has three sources of danger—(1) suffocation, (2) aspiration pneumonia, and (3) extension of the inflammation to the connective tissue of the neck and mediastinum.

If the original site of the swelling be the *isthmus of the fauces*, the first glance will suffice to distinguish between *diffuse* and *unilateral* **sore throat.** If it be diffuse, we must think of catarrhal and lacunar sore throat, scarlet fever, diphtheria, and secondary syphilis. As far as these are of surgical importance, they will be discussed later on with the subject of diphtheria.

The inexperienced often miss syphilitic sore throat when it appears in a mild catarrhal form, and is not accompanied by mucous patches on other parts of the mucous membrane. Those affected with "syphilis insontium" are of course unaware of its cause, and the others often enough refuse to know anything about it. But it is just in this stage that diagnosis is so important, both because of early treatment and the protection of the patient's surroundings.

If the sore throat is unilateral, we should entertain the possibility of a tonsillar or retro-tonsillar **abscess.** If the swelling increases, we should not wait till it bursts spontaneously, but should search for pus in the classical situation before complications supervene.

An acute swelling on the posterior or lateral wall of the pharynx must be regarded as an acute retropharyngeal abscess, which is generally due to a lymphadenitis, but in rare cases may depend upon osteomyelitis of the spine. If the abscess is not accessible externally we must evacuate it from the inside, using a puncture syringe so that the gush of pus should not inundate the larynx.

Further, we must not forget that there are certain acute inflammatory processes which lead to the growth of adenoids on the *roof of the pharynx.* The examination of this region is always important when children suffer from unexplained pyrexia.

If the *lips* and *cheeks* are infiltrated, and a diffuse swelling of the mucous membrane with severe turgidity of the gums is seen when the mouth is opened—as far as the lockjaw permits this—if there are already a few ulcers about, and fœtid froth issues from the mouth, we are confronted by the rare disease **gangrenous stomatitis,** which may be fatal in a few days, but the cause of which is still obscure. This disease exhibits, in the most intense degree, the symptoms present in scurvy and mercurial stomatitis.

If the inflammatory process is so severe that gangrene of the lips and cheeks ensues, it is impossible to miss the clinical picture of **noma,** which occasionally attacks children after debilitating infectious diseases.

CHAPTER XVIII.

TUMOURS AND ULCERS IN THE MOUTH, PHARYNX AND NOSE CAVITIES.

MANY new growths of mucous membrane appear as ulcers; we must therefore discuss them in common, when dealing with the oral and pharyngeal cavities. We have previously stated that *every obstinate ulcer must be suspected of malignancy,* however little aspect of growth it may possess. If this maxim were adequately taken to heart we should not so often see carcinoma treated for weeks with lunar caustic, until the enlargement of the glands of the neck finally takes the confirmed optimist by surprise, but renders the issue of an operation doubtful.

A.—NON-ULCERATING GROWTHS.

We only include here among " ulcerating growths " those wherein ulceration is of the essence of the disease, such as cancer, tubercle or syphilis, but not those which have been exposed to accidental superficial erosion, *e.g.*, an epulis wounded by an adjacent tooth. In cases of cancer it is often necessary to look for the ulcer. A cancer at the base of the tongue may start as a non-ulcerating growth, and only after a careful examination with a mirror or the palpating finger will a deep, open excavation be discovered posteriorly.

We shall proceed topographically, because the various diseases have their own special sites of preference.

(1) THE MUCOUS MEMBRANE OF THE LIPS AND CHEEKS.

If a patient complains of a little tumour about the size of a hazel nut, situated in the mucous membrane of the lip or cheek—a tumour which disappears and reappears, and examination reveals a bluish, translucent, semi-globular structure which is not dispelled by pressure, the diagnosis can only be a **mucous cyst**. But if the tumour in this situation is bluish-red, and is dispelled by pressure of the finger, the case can be nothing but one of **cavernous angioma**. It is noteworthy that the angiomata of the mucous membrane, in contrast to those of the skin, are distinctly encapsuled. A soft pedunculated growth, which cannot be dispelled by pressure, hanging from the mucous membrane of the cheek, is a **fibroma**, in which the various components of the mucous membrane, viz., glands, blood-vessels and lymphatics, are more or less extensively proliferated.

(2) THE FLOOR OF THE MOUTH.

The tumours in this region, covered by normal mucous membrane, are usually *cystic* structures, except for the very rare yellowish lobulated **lipomata** which are visible beneath the mucous membrane. The surgeon did not note the yellowish colour of the swelling, depicted in fig. 71, and he incised it, thinking it was a ranula.

A bluish tumour, rather laterally situated, shining through the thinned mucous membrane, soft, elastic, or fluctuating in consistence, and raising the tongue, is a **ranula** (fig. 69). This diagnosis states nothing as to the origin of the structure concerning which embryologists and surgeons are at considerable variance. The view at present prevailing explains most cases on the assumption that they arise as cysts from segments of the sublingual gland, much in the same way as the mucous cysts of the lips or cheeks, varying in size from a pea to a hazel nut, which we have just mentioned.

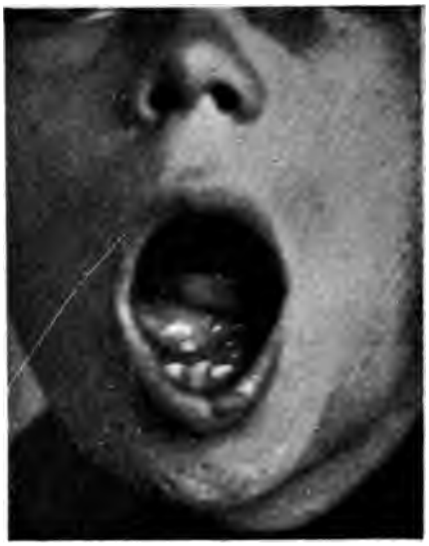


FIG. 69.—Ranula.

It has been shown that besides the ordinary ranulæ, cysts may arise in some cases from Bochdalek's ducts, which are lined with ciliated epithelium. Nuhn-Blandin's glands at the tip of the tongue have also produced cystic structures, but these have nothing in common with ranulæ, because they are situated at the tip of the tongue and not *under* it.

If careful attention is given to the various characteristics of ranulæ it is difficult to mistake them. *Lymphangiomata*, which have been observed in this vicinity, are much less sharply defined, and they frequently involve the tongue itself. They are composed of numerous small vesicles and do not constitute a single-spaced structure. *Lipomata* are lobular and appear yellowish, as seen through the mucous membrane. Only a **dermoid** could lead to error of diagnosis

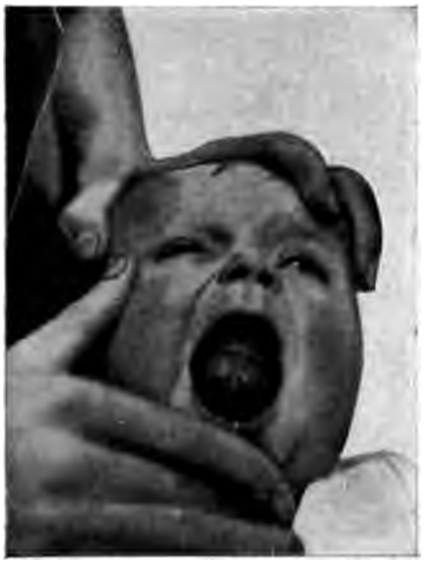


FIG. 70.—Sub-lingual dermoid.

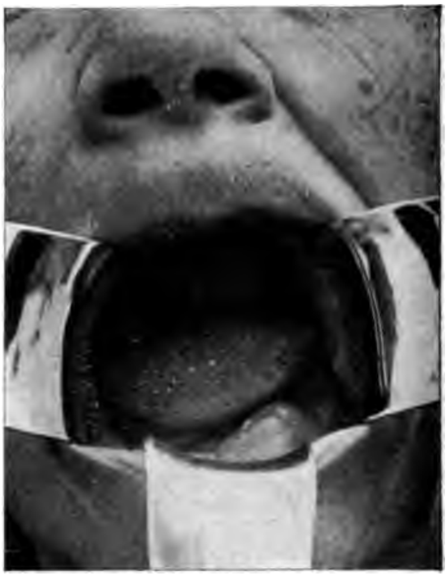


FIG. 71.—Lipoma of floor of mouth.

because it is also a single-spaced structure. Dermoids which occur in this region nearly as frequently as ranulæ, are, however, centrally situated, whereas ranulæ are rather laterally placed (fig. 70). Their walls are thicker than those of ranulæ, and the cysts present rather a whitish-yellow than a bluish appearance from beneath the mucous membrane. Sometimes they are closely adherent to adjacent tissues, or are even attached to the bone, which is never the case with ranulæ. They are also more liable to suppurate than the latter.

If doubt arises, because a tumour apparently in the median line gleams through the mucous membrane with a bluish tint, *e.g.*, a large ranula which has encroached on the middle line, or because a laterally situated cyst—dermoids are in rare cases lateral—looks like a dermoid, the diagnosis can be cleared up by a puncture, which is quite harmless. The treatment is similar in both conditions, so that an error in diagnosis has no serious consequences.

(3) THE GUMS.

A tumour projecting from the margin of the gum, varying in size from a pea to a walnut, may be summarily diagnosed as an **epulis,** *i.e.*, "a tumour of the gum." This is, of course, no histological diagnosis, it is merely a description of what is found. If the tumour is of the same colour as the healthy gum, and is firm, it may be

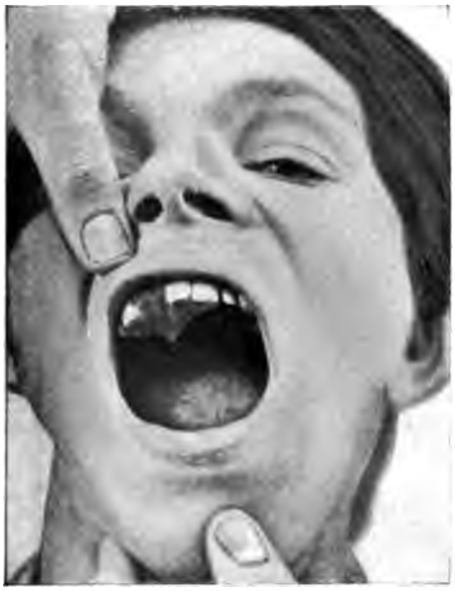


FIG. 72.—Epulis growing from a space between teeth.

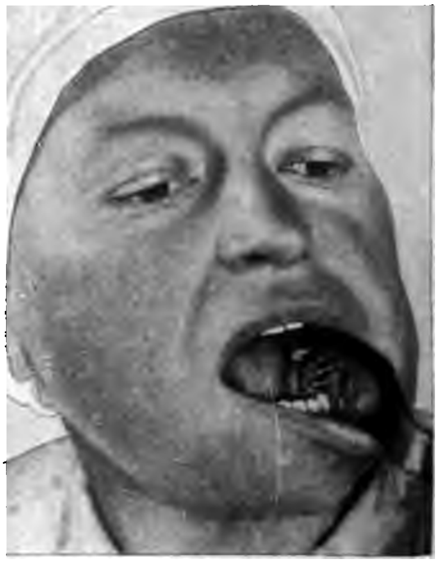


FIG. 73.—Epulis (pure fibroma), with impressions of upper molars.

regarded as a *pure fibroma;* if, with the same colour, it is softer, we must conclude that it is richer in cells and blood-vessels, and therefore approximates to a *sarcoma.* If the growth is darker, with a faint shade of brown, the case is one of *giant-celled sarcoma,* springing from the alveolar periosteum, the typical form of epulis.

These growths have an abundance of vessels on the surface almost like an angioma, all the rest of the tumour showing the structure of a giant-celled sarcoma. They frequently contain a brown pigment in addition. Their malignancy is limited to local occurrence; the glands are not affected nor do metastases occur.

It is noteworthy that an epulis will often grow from a site which has been exposed to persistent irritation, *e.g.*, in the spaces between teeth, in the neighbourhood of old stumps (fig. 72). Occasionally the epulis becomes somewhat ulcerated from friction on its surface, and it may even show the impression of the opposite teeth (fig. 73).

They can be promptly distinguished from tumours of the jaw proper by the narrow peduncle which connects them with their site of origin. At first sight they seem to be situated on a broad base, but if they are well elevated it is astonishing to see how slight their attachment really is.

Granulations, which occur so frequently in connection with remains of stumps, and not infrequently in pulp cavities, must be distinguished from epulides, which are really genuine tumours. In neglected teeth, the whole row of missing crowns may be occupied by such granulations.

(4) PALATE, TONSILLAR REGION, BASE OF TONGUE.

A semi-globular swelling which has recently developed in the middle of the hard palate is usually a *gumma;* but if it embraces the margin of the palatal plate, or if it is close to a diseased tooth or an old stump, it is a dental abscess (fig. 74).

If the growth arise from the *soft palate* or the *tonsillar region,* and is definitely capsulated with the mucous membrane movable over it, we must think of one of those mixed tumours which more frequently occur in the parotid region. If the tumour has a wide attachment to the tonsillar region, and is only slightly or not at all movable, the case is certainly one of sarcoma, and we must not wait for enlarged glands to confirm the malignancy. A striking swelling of the whole of the lymphatic system of the pharynx should suggest the possibility of leukæmia or pseudo-leukæmia.

The fact that many tonsillar sarcomata have vanished after energetic treatment with arsenic, and also with X-rays, has not only a therapeutic but also a diagnostic interest. These tumours must be classified with the still somewhat enigmatic group of lymphosarcomata.

Pedunculated polypi are sometimes found on the palatine arch, especially in the form of—purely innocent—papillomata.

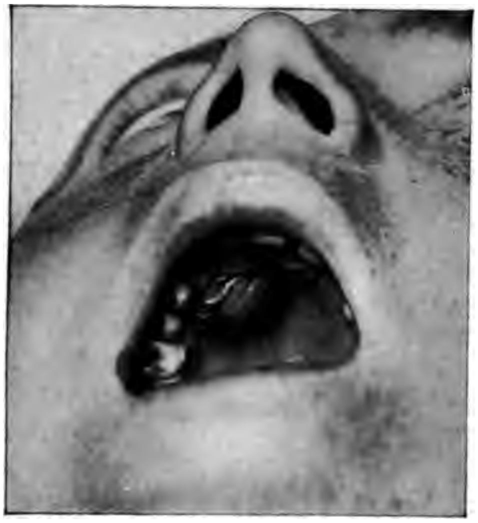


FIG. 74.—Abscess of palate arising from root of tooth.

A soft, roundish well-defined tumour at the base of the tongue is most likely to be an **aberrant goitre.**

This localization is explained by the circumstance that the central thyroid gland rudiment grows from the position which subsequently forms the base of the tongue. For this reason, accessory goitres may be met with along the whole remaining tract of this rudiment, between the foramen cæcum and the processus pyramidalis of the thyroid gland, *i.e.*, the thyroglossal duct.

Patients with lingual goitre feel as if they have a lump in the throat, which cannot get down. If the growth is extensive the speech becomes nasal, and ultimately respiration is obstructed. Sometimes severe hæmorrhage occurs from the superficial vessels.

(5) PHARYNX.

If a tumour-like structure projects into the pharyngeal cavity, one must endeavour to ascertain its point of origin, by means of the eye, the probe and the finger. If it has a broad attachment to the anterior surface of the vertebral column it might be a **sarcoma**, but it is more likely to be a tubercular **abscess** due to spinal caries. The stiff manner in which the patient holds his head will already have suggested this diagnosis to the careful observer ; and the history will show that the tumour in the pharynx was long preceded by difficulty in moving the head. Palpation will also show at once whether we are dealing with an abscess or a solid tumour; the latter may be simulated by retro-pharyngeal projections of tubercular glands.

If the tumour projects into the pharynx from above, it may be an ordinary **mucous polypus** arising from the nose. These are remarkably soft to the touch, and they give the impression of receding out of the way of the palpating finger. If they are visible, directly or through the mirror, they are recognized by their well-known bluish colour and their glassy translucent appearance. If the growth is of firmer consistence we may be undecided as between a fibroma springing from the basilar fibrocartilage at the base of the skull in young people, and a sarcoma proper of the base. As the differential diagnosis between these two is of extreme importance for prognosis and treatment, we must dwell upon it for a moment.

A fibroma of the base of the skull, usually a **nasopharyngeal fibroma** or a nasopharyngeal polypus, can be excluded if the growth has occurred *after* the termination of adolescence, *i.e.*, after the second decade. Naso-pharyngeal fibromata have the peculiarity of only developing before the termination of this period. But if the patient has not yet completed adolescence the growth might be of either variety. In doubtful cases, the sex as well as the age is of assistance, because in infancy nasopharyngeal fibromata are of equal incidence in both sexes, whereas at puberty, when they are most frequent, they only attack males. The previous history is also of great importance. If the growth has taken years to develop and has manifested itself by nasal obstruction and occasional severe hæmorrhages, we can definitely exclude sarcoma. On the other hand, if the existence of the growth, the blocking up of the posterior nares, and possibly also hæmorrhages, have all taken place within a few months, we must regard the case as one of sarcoma. If the new growth emits processes into all the accessible cavities in the neighbourhood, processes which, if visible, display the same roundish form, and the same sharp definition as the tumour within the pharynx, the case is one of fibroma. On the other hand, a diffuse extension of the tumour, the early onset of neuralgic pains and brain symptoms are in favour of sarcoma.

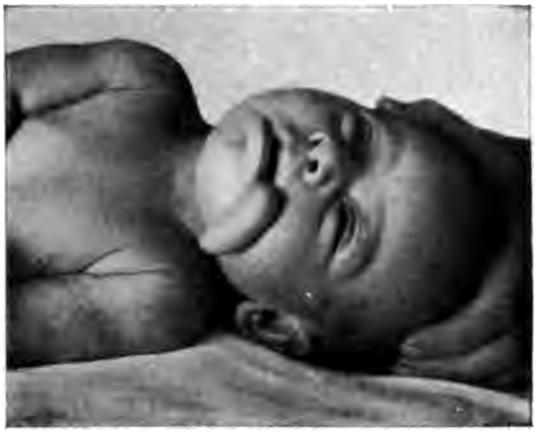


FIG. 75.—Pharyngeal polypus, with a long pedicle arising from the palatopharyngeal arch.

Teratoid growths of various kinds are to be found at the junction of the pharynx and œsophagus as well as in other positions in the pharynx. They may be teratomata proper formed from all the three layers of the embryo, with a tuft of hair on the surface, or they may be simple *lipomata* or soft *fibromata;* the latter may at times hang out of the mouth like a sausage (see fig. 75). At other times they only appear after some definite cause, such as vomiting, and then are again swallowed by the patient.

(6) THE NASAL CAVITY.

Non-ulcerating new growths in this region, as in the pharynx, may be **mucous polypi, fibrous nasopharyngeal polypi,** or **sarcomata.** We have already discussed the first two, and would only add here there is usually some accessory sinus catarrh behind the mucous polypi. But for the formation of the polypi there is also required a special, and often a very obstinate predisposition, which tends to the constant development of new polypi. In fact, the shape of the nose may be so greatly deformed in some cases in the course of a few years, that a fibrous polypus or even a sarcoma would be diagnosed, if the previous history were unknown. Sometimes the appearance of a bluish mucous polypus at the nostril leads immediately to a correct diagnosis.

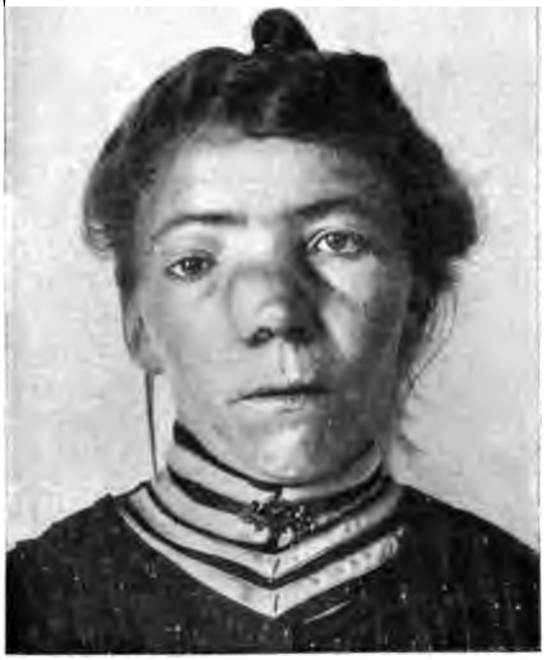


Fig. 76.—Mucous polypus of the nose.

This figure represents a patient who regularly for fifteen years was relieved from time to time of whole bunches of mucous polypi from both nasal passages, before she made up her mind to undergo operative treatment for her bilateral accessory sinus catarrh.

Sarcomata of the nose usually start from the turbinated bones, and in the beginning are merely regarded as hypertrophied membrane. When they take on rapid growth, cause nasal obstruction and hæmorrhage, a small portion excised for histological examination will make the diagnosis positive, or confirm it if already made.

B.—ULCERATION PROCESSES.

Two points must be impressed upon the beginner in regard to the diagnosis of ulcers, viz., to closely examine the characteristics of the margin of the ulcer, and to investigate its base. This rule also applies to ulcers of the oral cavity, but it encounters many difficulties in this region owing to anatomical conditions and the occasional inaccessibility of the tumour. However, a superficial ulcer with a soft border and a soft greyish base is *tubercle;* an ulcer with a fatty, yellowish base is a *gumma:* a hard undermined border with a hard segmented base, often covered with necrotic shreds, points to *cancer;* and an ulcer of firm consistence without undermined edges, but with a smooth, varnished-looking base suggests a **primary chancre.** But one must not conclude from this that a cancer must always have undermined edges and be segmented, and that a gumma necessarily must have a yellowish, fatty base in all its stages.

Our diagnosis will always be most accurate when we bear in mind the most frequent morbid conditions which occur in the various portions of the oral cavity.

(1) THE MUCOUS MEMBRANE OF THE LIPS AND CHEEKS.

Cancer is the most frequent lesion in this part, although tubercle, gumma, and primary chancre are possibilities. We have already discussed cancer of the lip. Cancer of the mucous membrane of the lip is much rarer, but its prognosis is much worse.

The following case shows how difficult the diagnosis may be, on the assumption that an ulcer of the lip, in an old man, must be cancerous.

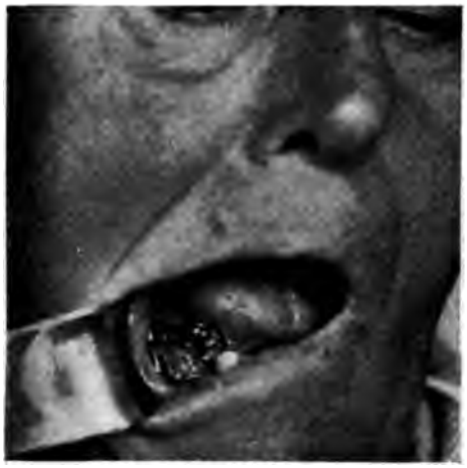


FIG. 77.—Sub-lingual cancer.

A man, over 70, an old sufferer from bronchitis, had an ulcer on his left lower lip, and another one on the mucous membrane of his right cheek. They were both soft, superficial and very painful. There were neither epithelial plugs nor tubercles visible. The history of syphilis was very indefinite, and the pain contraindicated it. The only point in favour of cancer was the age, everything else was against it, and the pain suggested tubercle. The bronchitis appeared to be an ordinary senile bronchitis. The diagnosis of tubercle could only be made by a process of exclusion, but its accuracy was established by histological examination of a piece of the border excised for the purpose.

It is hardly necessary to mention the small transitory ulcers on the mucous membrane of the cheek, which are so often produced by *bites*.

(2) THE FLOOR OF THE MOUTH.

Carcinoma is of frequent occurrence on the floor of the mouth; tubercle and primary chancre are rare. In its early stages the cancer appears as a small, movable, roundish, definitely raised tumour, presenting at its centre a small superficial ulcer surrounded by an encroaching border. This in itself suffices to justify the diagnosis. The practised observer will involuntarily feel for enlarged hard glands after examining the growth in question, in order to confirm the diagnosis. The presence or absence of glands must, however, not be invested with too great a significance; at any rate their absence is not a conclusive argument *against* cancer. If the tumour has become adherent to the jaw and eventually also to the tongue, so that the latter gets fixed and movements of mastication and speech are interfered with, the diagnosis is easy enough.

(3) THE GUMS.

The careful examination of the gums is not only of importance to the dentist and the physician, but also to the surgeon. For instance, a persistent colic, which might be attributed to organic obstruction of the bowel, will be shown to be toxic in origin, by the discovery of the well-known blue line on the gums.

In a case of obstinate *suppuration* between a tooth and the gum, and general loosening of the teeth, we should not treat the gum, but should examine the urine for sugar. If this examination does not reveal the cause of the *alveolar pyorrhœa*, some other general disease should be searched for.

If called to arrest obstinate hæmorrhage from a tooth, we must not be content with applying a styptic, but must investigate the cause of the bleeding, which may be due to a hitherto overlooked *hæmophilia*, to a *leukæmia*, or to chronic jaundice.

Most *chronic inflammatory diseases* of the gums depend upon dental disease. If they do not recover after extraction of the bad teeth, the removal of stumps or sequestra, we should suspect actinomycosis, tubercle, or phosphorus necrosis. We have already discussed the diagnosis of these conditions.

Ulcers which are not surrounded by a definitely inflamed area must be diagnosed in the same way as those on any other part of the oral mucous membrane. It will be necessary to differentiate between *cancer*, *tubercle*, and *gumma*. *Primary chancre* is very rare. The leading points of differential diagnosis have already been adequately stated.

Finally, swellings which bleed easily are due to scurvy, and in little children indicate Barlow's disease.

It is noteworthy that in the latter condition the unmistakable bluish-red swelling of the gum only occurs where teeth have already erupted. The pain in the extremities completes the clinical picture and demands that we should not incise the swelling in the gum, but should adopt immediately the only prompt and effective treatment, *i.e.*, the abandonment of all artificial or artificially sterilized food.

(4) THE TONSILLAR REGION.

Various forms of ulcer, cancer, primary chancre, tubercle and gumma have to be considered here, as well as the somewhat rare non-specific ulcer of the tonsil. The first lead in diagnosis is given by the presence or absence of *glandular enlargement*.

(*a*) If the glands are not enlarged we may exclude chancre, unless it be quite recent; and we should think of cancer and gumma, possibly also of tubercle.

The frequency of **carcinoma** renders its presence more likely than that of any other ulcer. If the patient is an alcoholic, this supports the diagnosis, because, in my experience, tonsillar cancer, and especially cancer of the pharynx, have mainly occurred among heavy drinkers. The fact that only one ulcer is present is also in favour of cancer, apart from its hard base and border. Gummatous and tubercular ulcers are, on the other hand, frequently multiple. Pains radiating towards the ear and robbing the patient of sleep at once dispose of gumma and tubercle. Pain limited to swallowing is strong presumption against a gumma, but not against tubercle. The absence of pain is, however, no evidence against cancer, because pain may not come on until a stage wherein operative measures are useless.

If the consistence and, perhaps, the multiplicity of the ulcers show that it is either **tubercle** or **gumma**, the presence of separate little nodules on a reddened base around the ulcer points to tubercle, whereas the partial aggregation of areas which start as roundish nodules and their subsequent disintegration in the centre, point to gumma. These characteristics are sometimes difficult to detect, and it is therefore all the more important to pay attention to the *history* of the patient and his *general condition*. There is generally some preceding pulmonary or intestinal tuberculosis in cases of pharyngeal tubercle, and although the most carefully taken history will not always reveal syphilis in cases of gummata, it very frequently will do so. In doubtful cases, we must resort to *histological* and *bacteriologica* investigation, lest we overlook cancer.

A small piece of the edge of the ulcer should be snipped off with forceps, or scissors, and a part thereof submitted for histological examination and a part used for animal inoculation. The microscope will settle the diagnosis with certainty in a few hours or within a day or two; guinea-pig inoculations take at least four to six weeks to supply definite information. If this investigation is negative and clinical signs are suspicious, we must not be content until another and larger piece has been removed, or a piece from the margin or base of the ulcer, if it is impossible to establish the diagnosis in any other way.

The result of a Wassermann test should always be taken into consideration, but it must not be forgotten that a syphilitic may become affected with tubercle or cancer. Diagnosis "ex-juvantibus" still holds the fields in syphilis.

(*b*) Diagnosis is facilitated if *enlarged glands are present.* If they have come on soon after the appearance of the ulcer, and have reached a fair size in a short time, and are adherent to the adjacent tissues, we may assume that the glands are in a state of early fibrosis. The minutest superficial injuries suffice for infective material derived from dirty drinking vessels to stick to the tonsils.

One illustration, out of many, may be given. A student drank out of a drinking horn directly after an old man, in token of mutual friendship. The student acquired a tonsillar chancre, and it subsequently transpired that the old man had signs of secondary syphilis in his mouth—an instance, by the way, of the neglect of elementary rules of hygiene, prevalent at the present day.

If the glands have not enlarged until some considerable time has elapsed since the appearance of the ulcer, then the diagnosis lies between carcinoma and tubercle. In the former case they are hard, in the latter rather softer. In both cases they may become adherent, so that no conclusion can be drawn from this condition; but enlarged glands which have existed for many months without contracting adhesions around are more likely to be tubercular than cancerous. The adhesions of cancerous glands are of a very firm kind, so that hard immovable masses are formed. After tubercular glands have contracted adhesions, suppuration generally takes place in the centre, so that it will be found that soft, elastic, and even fluctuating areas exist, surrounded by comparatively hard borders.

No conclusions can be drawn from the condition of the ulcer, or of the glands existing at the time, if the attention of the patient or the medical attendant has first been directed to the presence of an ulcer by the onset of glandular enlargement.

It may be stated finally that Plaut-Vincent's angina (*see infra*) in the ulcerative stage may easily be mistaken for a syphilitic ulcer. The bacteriological findings and the rapid recovery are decisive.

(5) HARD AND SOFT PALATE.

A solitary ulcer spreading from the *tonsillar region* towards the margin of the soft palate is usually a **carcinoma.** Carcinoma rarely begins on the soft palate, but we have seen it entirely eaten away by a primary cancer. A swelling in this region is much more likely to be a **gumma,** especially if it, or the ulcers, are near the *hard palate,* or actually on it, or if they have already perforated it.

Tubercle also occurs on the soft palate. It differs from gumma in its longer duration, in the appearance of the ulcer, and also by causing severe dysphagia, and by rarely failing to produce glandular enlargement.

(6) PHARYNGEAL WALL.

Ulcers on the mucous membrane of the pharynx are of rare occurrence, apart from those at the base of the tongue and the tonsillar region. **Cancers** are usually found at the entrance to the gullet, in the neighbourhood of the roof of the pharynx and in the vicinity of the posterior nares. As they arise in such concealed situations, enlargement of the glands is usually their first indication, and they require a careful rhinoscopic examination. Ulcers on the posterior wall of the pharynx are usually **gummata.** It is also necessary to mention the **bedsore** caused by the cricoid cartilage in very chronic diseases.

(7) NASAL CAVITY.

We leave the round ulcer of the septum to the rhinologist, so that cancer, syphilis and tubercle again come under consideration. A careful examination with a mirror is generally indispensable, but there are certain concomitant conditions of the diseases which are useful for differentiating one from the other.

There are certain tumours and ulcers of the pharynx and nose, which would only be diagnosed if we are aware of the patient's occupation, or of his geographical relationships. These include the **ulcers of glanders** in the nose of people who attend to horses suffering from farcy, **leprosy,** and **rhinoscleroma** of certain districts, the latter usually from the Balkans. The diagnosis of leprosy will be made from the other appearances of the disease, and rhinoscleroma will be recognized by the hardness of the infiltration, the absence of glandular enlargement, and the chronic course of the disease.

A young man returned home from a sanatorium with his pulmonary condition improved, but he had an ulcer in the nose which gradually ate away the septum. In such a case, nothing but *tubercle* would be thought of.

An aged grandmother complained of "a cold in the head," or, rather, of a persistent and profuse nasal discharge. She produced a few shreds of bone, which, to her amazement, had escaped with the discharge. Her previous history told of one living child after a series of miscarriages. Iodide of potassium worked wonders. We shared her pleasure at this result, but we were careful not to tell her that this was a reminder of her late husband, whose portrait discreetly smiled on us from the wall.

A middle-aged patient came with a bloody, offensive discharge from one nostril, which had lasted for some months. There had been hard glands at the angle of the jaw for the last few weeks. This could be nothing but cancer.

CHAPTER XIX.

CHRONIC DISEASES OF THE TONGUE.

THE custom of the old physicians—who invariably looked at the patient's tongue—was no idle habit, and the younger generation neglect this diagnostic aid too much, in favour of "exact" methods of diagnosis. The surgeon should find interest not only in the colour of and the deposit on, the tongue, but particularly in its degree of moisture. Nothing affords us a more rapid conception of the patient's condition and of the prognosis in infective diseases, like peritonitis, than a glance at the tongue. A dry tongue of normal colour is worse than a moist tongue, however much coated it may be.

We will not refer to the various superficial changes, such as the map-like tongue, the black, hairy tongue, the wrinkled tongue, &c., because they possess no surgical significance; but leukoplakia must be noted, because it forms an excellent soil for the development of cancer. It is now generally recognized that the abuse of tobacco and syphilis are equally responsible for this latter condition.

Fournier has asserted that in Paris cancer of the tongue is, so to say, a sequela of syphilis, with leukoplakia as an intermediate stage. This statement can only be applied to other countries with considerable reserve.

As in the preceding chapter, we must separate here also the ulcerated from the non-ulcerated changes.

(1) NON-ULCERATED TUMOURS AND SWELLINGS.

We must first refer to **macroglossia** among the new growths of the tongue. It is a diffuse enlargement of the whole organ, which does not concern so much the musculature which is comparatively unaffected in macroglossia, but consists of an increase of interstitial tissue and lymph spaces, so that it might, with more or less accuracy, be called a *diffuse lymphangioma*. When this condition is fully developed it imparts an imbecile expression to the face, but we must not therefore conclude that all who are affected therewith are idiots. Nevertheless, macroglossia does occur most frequently amongst those whose mental development is defective. One of the signs of hypo- and athyroidism is a certain degree of diffuse enlargement of the tongue, which gradually decreases in size under the influence of specific treatment.

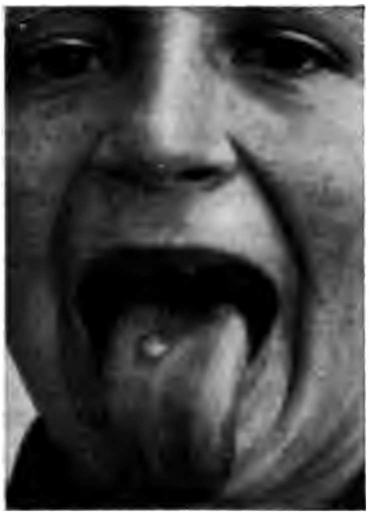


FIG. 78.—Lymphangioma of the tongue.

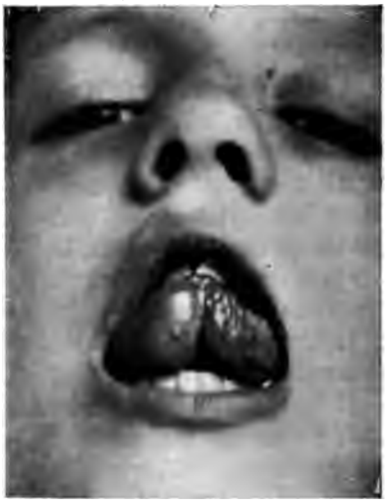


FIG. 79.—Cystic lymphangioma of the tongue.

Among the *true tumours of the tongue* **cavernous angioma** is at once recognized by its colour and by easily emptying on pressure. Circumscribed lymphangiomata are not so easily diagnosed. They appear as fairly firm nodules in the soft tissue of the tongue, usually on the dorsum (fig. 78), but sometimes also on its under surface (fig. 79). As with other lymphangiomata, their contents cannot be well expressed. Were it not for the long duration of the disease, one might be tempted to think of some chronic inflammatory process of a tubercular nodule, or of actinomycosis. But if we carefully examine the tongue we shall note that the papillæ around the tumour are prominent and enlarged and that some of them form little vesicles. This is decisive for the diagnosis of lymphangioma.

If we feel soft lobulated growth within the substance of the tongue we may claim it as a **lipoma**, and thus add one more to the dozen cases which have hitherto been recorded.

But much more important than the recognition of such rarities is the accurate diagnosis of *tubercle, gumma, actinomycosis, sarcoma* and *cancer*.

We mention cancer last, and then only with considerable reserve, because it never occurs in the tongue with an undamaged mucous membrane. Glandular cancer, which alone can come into consideration, is so rare that it is hardly necessary to reckon with it. But, on the other hand, inexperienced observers are liable to declare that a cancer is not ulcerated when they have failed to see it properly. Careful observation will often reveal a deep ulceration below the raised mass of new growth which may be covered by normal mucous membrane, and close inspection will often show that a nodule which can be easily seen and is apparently non-ulcerated is covered by epithelium which even macroscopically is abnormal,

A *pedunculated nodule*, soft as the tongue itself and situated thereon, must be regarded as a **fibroma**. It is rare, but occurs on the tongue, as it does on other parts of the oral mucous membrane. Its long duration will serve to prevent its confusion with sarcoma. A nodule which has arisen within a few months and is firmer than the tongue substance must, however, be looked upon as a **sarcoma**. There are also soft sarcomata; but they do not possess the toughness of soft fibromata; they soon break down and form deep ulcers.

If the nodule is *infiltrated* within the tissue of the tongue we must differentiate between *tubercle, gumma, actinomycosis* and *sarcoma*.

It must be regarded as a **sarcoma** if its size is greater than is compatible with an inflammatory granulation tumour. A lump as large as a hen's egg will be neither a tubercle nor a gumma. In the case of smaller tumours the early onset of radiating pain points to malignancy. But, as a rule, we should only diagnose sarcoma of the tongue when there is no more plausible possibility, because it is a condition of great rarity.

The local circumstances often suffice to differentiate between **gumma** and **tubercle**. A nodule of a few weeks' standing which has not yet broken down, is more likely to be tubercle than gumma. Other things being equal, the fact that a tumour is single is in favour of tubercle. Tubercular nodules are also more painful than gummata; the latter may be sensitive on pressure but are only very slightly painful in themselves. Soft swelling of the glands of the neck points to tubercle, as already stated in connection with ulcers of the pharynx. Gummata are not associated with glandular enlargement, if they are unbroken and therefore not secondarily infected. The previous history and general condition of the patient are also of importance. Experience shows that tubercle of the tongue is very rarely a primary manifestation; as a rule, it occurs only in patients who are suffering from pulmonary or abdominal tuberculosis. Similarly, in cases of gumma, the history or the general condition of the patient will provide evidence of old syphilis. Finally, as in all these cases, we have the serum reaction and the therapeutic test as ultimate resources.

A deep-seated **nodule of actinomycosis,** which occasionally occurs in the tongue, is distinguished from gumma by its hardness, and from tubercle by the absence of the characteristic glandular enlargement. If the disease has involved the surface it consists of a board-like infiltration of the tongue permeated by soft foci of granulation tissue, so distinctive of actinomycosis elsewhere.

(2) ULCERATIVE DISEASES OF THE TONGUE.

In coming to any conclusion about an ulcer, it must not be forgotten that all lesions within the oral cavity tend to ulcerate, especially as a consequence of friction against the teeth. The mucous membrane over a sarcoma may therefore become destroyed from secondary causes. But in such a case the growth itself is usually so prominent that the true state of affairs is perfectly evident. Advanced sarcomata, which have broken down extensively, are easily mistaken for cancer, in the absence of a microscopic examination.

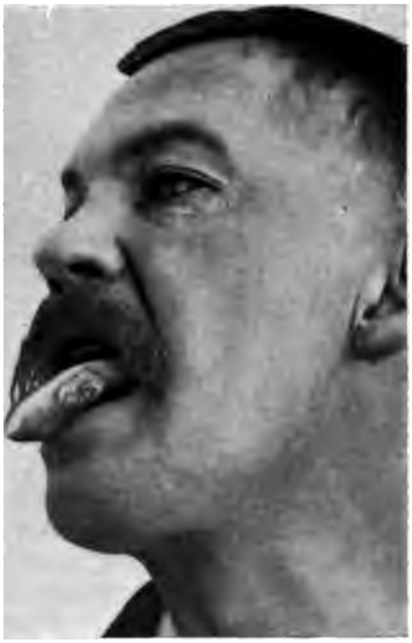


FIG. 80.—Cancer at the edge of the tongue.

The reader is referred to the discussion on ulcers of the oral cavity for the differentiation between *tubercle, primary sore, gumma,* and *cancer,* but a few special points may be mentioned here.

When a deep-lying **tubercular** focus breaks down the lesion in the mucous membrane does not always assume the characters of a fully developed ulcer. A probe only penetrates through a small opening, more like a fistula than an ulcer, into a wide pocket corresponding to the original extent of the diseased focus. But when a gumma breaks down there forms immediately an extensive lesion of the connective tissue and mucous membrane. Gummata differ from cancer in being situated in the middle of the dorsum of the tongue or on its tip. Cancer nearly always starts on the edge of the tongue (fig. 80) and gradually invades the centre.

Besides the well-defined gummata of the tongue there occurs occasionally a *diffuse gummatous infiltration* of the organ, but this is differentiated from cancer by its indefinite limitation and the fact that it does not break down (fig. 81).

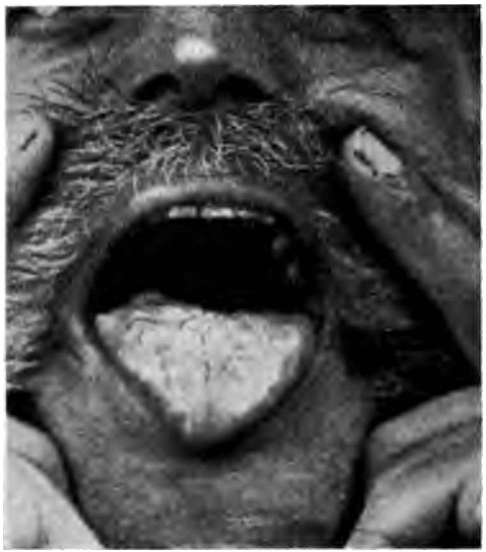


FIG. 81.—Gummatous glossitis.

Finally, it should be noted that deep and often painful *rhagades* on the tongue are to be attributed to tertiary syphilis, although this may be the only sign of the disease.

As far as the diagnosis of **cancer** is concerned the warning must again be given that it is negligent to await the development of all possible symptoms and finally glandular enlargement, before informing the patient of the nature and gravity of his disease. It is especially wrong to comfort him with the assumption that he is suffering from an *ulcer caused by a tooth*, until it becomes too late. Often enough it does happen that a sharp tooth or a jagged stump injures the edge of the tongue, causing a small superficial ulcer which fails to heal because it is subject to constant friction. But the removal of the offending tooth or the filing of the sharp points, without doing anything at all to the ulcer, will result in its healing within a few days. If it does not heal it signifies that the case is somewhat more serious, and careful palpation will no doubt reveal distinct hardness of the base and margin.

But cancer does not always begin in this manner. Sometimes it starts as a small hard nodule, with no loss of surface epithelium visible to the naked eye, but around which slight contractures of the tongue tissue may be seen. Such a condition renders any further observation superfluous ; it demands immediate operation. In other cases we have to deal with patients who have been suffering from leukoplakia for years. Having been informed that this disorder predisposes to cancer they bestow the necessary, and more than necessary, notice on their oral mucous membrane, and obtain medical advice as soon as they discover any thickening of a leukoplakia patch. Often enough such a patient is obsessed with the fear of carcinoma, and fancies that there is some hardening present when in reality there is none. The practitioner must not be too ready with his reassurance, the suspected spot must be most carefully palpated and compared with the other spots. If there is any hardening it must be treated as cancer, even though the unaided eye detects no loss of epithelium. At any rate the patch should be excised and submitted to microscopic examination, with a view to a more extensive operation if the diagnosis is confirmed.

Cancers, which so frequently start at the hinder edge of the tongue, opposite the tonsil, escape detection for a considerable time. When they are discovered they are usually so far advanced that it is impossible to say whether they have started from the tongue and invaded the tonsil or *vice versa*.

It is usually said that cancer of the tongue occurs in middle and in old age, and only in the male sex. But such a generalization should not be allowed to influence us too much in arriving at a decision. This is illustrated by the following case.

A girl, aged 22, consulted a doctor about an ulcer on the side of the tongue. The age and sex were such strong contra-indications of cancer that the treatment was limited for a long time to gargles and the application of lunar caustic. Eventually, the doctor became uncomfortable at the constant increase of the ulcer. Examination then showed that a large portion of the left border of the tongue was occupied by a shallow ulcer with slightly projecting but somewhat undermined edges. The border and the base were hard, but only to a slight depth. There were a few hard glands in the neck. There were no points in favour of tubercle or syphilis. The clinical diagnosis of cancer was confirmed by an exploratory excision. The operation, which was very extensive, did not succeed in preventing a recurrence.

PART II.

SURGICAL DISEASES OF THE NECK.

CHAPTER XX.

SURGICAL DISEASES OF THE RESPIRATORY TRACT (PHARYNX, LARYNX, AND TRACHEA).

DISEASES of the larynx have become so separate a department of practice that the general practitioner, in the stress of his daily work, does not scruple to declare himself unequal to them, at any rate he consoles himself for not making a careful examination by avowing that he is not a specialist. Although it is true that the diagnosis of these diseases at the present day demands complicated methods which are often out of the reach of the general practitioner, there still remain a number of maladies which he can and must correctly diagnose. It may then be necessary to refer the patient to a laryngologist for confirmation of the opinion or for treatment. It is not to be expected that the general practitioner should be familiar with such new apparatus as the tracheoscope and the bronchoscope, but we may anticipate that he is capable of rendering the larynx accessible by means of the laryngoscope and perhaps also with Kirstein's spatula.

The symptomatology of laryngeal diseases is very simple, comprising hoarseness, dyspnœa and difficulty in swallowing. This very simplicity explains the impossibility of making a diagnosis without the laryngoscope, unless the history and extra-laryngeal signs declare the nature of the disease. The examination should invariably be concluded with the laryngoscope, but we should always ascertain as much as we can without it.

We will take the cases as they occur in practice.

A.—ACUTE DISEASES.

(1) INFLAMMATORY PROCESSES IN THE PHARYNX AND LARYNX.

Our first question must be directed to the manner of onset of the laryngeal symptoms. If they have been preceded for a few days, or even only for a few hours, by a general feeling of malaise, if they began with difficulty in swallowing and culminated in hoarseness and dyspnœa, we shall at once suspect an acute infectious disease—a **pharyngeal and laryngeal diphtheria.** The younger the patient the stronger will our suspicion be, although it must not be forgotten that this disease also occurs among adults.

The *temperature* affords no decided clue. Many a slight case, with a trifling rise in temperature, has rapidly developed laryngeal obstruction. Indeed, I would go further and say that mild fever with severe local symptoms distinctly indicate diphtheria, because, other things being equal, Löffler's bacilli do not raise the temperature as much as streptococci do.

It should be noted incidentally that in the very severe cases the temperature falls to the degree of collapse, after a preceding pyrexia. The gravity of the general condition will lead to a correct conclusion.

The *examination of the pharynx* will supply further information. If we find the familiar thick whitish deposit upon the tonsils, fauces and even on the posterior pharyngeal wall—a deposit which can be removed as shreds—we diagnose diphtheria, at least clinically.

I have nevertheless seen deposits in streptococcal infection which could hardly be distinguished clinically from those of true diphtheria; but these cases are not frequent. They occur mostly in scarlet fever. The deposits in streptococcal sore-throat are usually less coherent, and are more easily rubbed off than those due to Löffler's bacilli.

If there be merely white specks on swollen red tonsils we must decide whether they are merely plugs of pus situated within the crypts of the tonsils—**follicular tonsillitis,** or whether they are accumulations *on* the mucous membrane of the tonsils. Only the latter are or can be diphtheritic. I say "can be," because streptococci may mislead us here also. But the indispensable bacteriological examination will show whether diphtheria is present—often a simple streak preparation suffices.

If the tonsils merely present a diffuse redness without any white specks at all, the case is most probably one of ordinary catarrhal tonsillitis, but diphtheria is by no means excluded. This cannot be absolutely excluded even when nothing has been found in the throat. There is only *one* disease which occasions parents unnecessary alarm—viz., **false croup.** A child who has been running about the whole day in good health, becomes suddenly ill at night with a hacking cough and symptoms of dyspnœa, coming on in paroxysms. On examination, the temperature is normal or only slightly raised, the throat is somewhat reddened, and between the seizures the general condition seems to be good. A moist compress round the throat, the inhalation of steam and a mild sedative suffice to banish all the trouble by the morning. True diphtheria seldom sets in so rapidly, and never departs so quickly.

The foregoing leads to the conclusion that every case of persistent dyspnœa, however mild, associated with general malaise, must be considered as serious. If there are at the same time symptoms of faucial diphtheria, they will confirm our diagnosis of laryngeal diphtheria; but their absence does not exclude this diagnosis.

There are a few *secondary symptoms* which have not yet been mentioned but which deserve consideration. The chief one of these is the *enlargement of the glands* of the neck. The absence of this enlargement is not evidence against diphtheria, but if it is present the infection is a severe one and we cannot allow ourselves to be consoled with the idea of a false croup. The glands do not indicate the nature of the infection, although they enlarge more frequently in diphtheria than in streptococcal sore throat. The same remarks apply to *splenic enlargement* and to *albuminuria.*

It is obvious from the foregoing that to confuse diphtheria and streptococcal laryngitis is not only pardonable but is often unavoidable. But there are mistakes which should be avoided. More than once has an incomplete history failed to elicit the presence of a foreign body, and a diagnosis of "diphtheria" has been made. In the absence of an epidemic every so-called case of "croup" which has come on suddenly without any prodroma, should remind one of the possibility of a foreign body and the history should be completely investigated from this point of view.

Still more erroneous is its confusion with **pneumonia.**

A little child was brought to the hospital in a state of severe dyspnœa. His temperature was high, and he was evidently very ill. A young assistant, who was much struck by the dyspnœa, forthwith reported the case as one of croup requiring tracheotomy. He did not notice that the child was breathing rapidly without any stridor. A more careful examination showed that the dyspnœa was due to extensive pneumonia.

The beginner should note that obstruction of the upper respiratory tract *slows* the breathing; diminution of the respiratory surface as in pneumonia *accelerates* the breathing. The reason for this is very simple. In order to allow the same amount of air to pass through a diminished transverse area of the respiratory tract, a prolongation and a deepening of the respiratory movements are required, at first at the expense of the respiratory pause. As the difficulty increases, more work is thrown upon the respiratory muscles, and there is more necessity for intervals of rest. The breathing, which at first was only deepened, becomes slowed as it increases in urgency. An accelerated and therewith an unavoidably weakened respiration can only suffice when the respiratory surface is diminished, as in pneumonia, but it would not be able to overcome a mechanical obstruction. When the muscles are fatigued, that is to say, in a state of asphyxia, there may be a relative acceleration of the breathing, even if there is mechanical obstruction, but it does not resemble the hurried respiration of pneumonia.

In addition to the frequency of respiration it is important to note the presence or absence of drawing-in of the soft parts of the thorax, the root of the neck, the supra-clavicular fossæ, the lower thoracic segment, and of the epigastrium. But this must not be confused with the so-called peri-pneumonic retraction of the lower border of the lung in young children.

It might appear to be quite superfluous to refer to these well-known matters; but I once saw a doctor blamed by parents for having submitted a child who was suffering from pneumonia to tracheotomy. Careful attention must therefore be paid to these signs in every doubtful case, and doubtful cases can occur in diphtheria. For instance, pneumonia may lead to dyspnœa in this disease, just as well as extension to the larynx may. It is therefore necessary to estimate to which of the two factors the dyspnœa is to be attributed. If it be due to pneumonia, we must await the effect of the serum; if, on the other hand, it is due to obstruction, we must operate despite the pneumonia. No sign is more conclusive than the type of the breathing.

The association of ideas by which laryngeal obstruction in children at once suggests diphtheria may lead to mistakes. The cause of the respiratory difficulty need not be either in the larynx or trachea, but the glottis may be blocked by a **retro-pharyngeal abscess.** Such an abscess is often tubercular, and starts either in the vertebral column or retro-pharyngeal tubercular glands. Non-tubercular retro-pharyngeal abscesses are usually the sequelæ of scarlet fever or measles, occasionally of erysipelas. If the pharynx is examined in every case of "croup" before proceeding to tracheotomy, these abscesses will not be overlooked.

Retro-pharyngeal abscesses, especially of the tubercular variety, obstruct the glottis by their own mass, but every acute inflammation in the neighbourhood of the larynx may produce a similar obstruction by exciting an inflammatory œdema of the entrance to the larynx, a so-called **œdema of the glottis,** or to be more correct,

laryngeal œdema. The loose submucous tissue may swell up so rapidly that the ary-epiglottic folds and the ventricular bands look like cushions, and a fatal result may ensue before assistance is forthcoming. Any infective process in the throat, acute abscesses, phlegmons or erysipelas may be responsible for this condition, or it may originate in a small wound caused by a pointed foreign body. Not a year passes without accounts of persons being suffocated before help is available, through swallowing a bee or a wasp with fruit or juice, and being stung by the insect. Œdema of the larynx may also occur after operative procedures in the neighbourhood of the throat; therefore patients who are threatened by this danger should be most carefully watched.

This brings us to the so-called **laryngeal perichondritis,** whose chief danger is the development of acute laryngeal œdema. This is not a primary disease, it is always the consequence of a *deep laryngeal ulcer* of some kind, or it may be a *metastatic process.* Such ulcers occur in infiltrating injuries, typhoid fever, small pox, tubercle, syphilis, cancer, &c., and suppurating metastases are met with in the course of typhoid, small pox, scarlet fever, and pyæmic diseases from any source.

If hoarseness and dyspnœa occur in any of these diseases, and there are at the same time external swelling and pain on pressure over the whole or part of the larynx, we must think of perichondritis. The laryngoscope will reveal the presence of ulcers, abscesses, and œdematous areas in very varied distribution.

(2) PURE CIRCULATORY DISTURBANCES.

Œdema of the larynx may exist, independently of any inflammatory disease, merely as a consequence of a *pure circulatory disturbance.* It may occur as a part of a general œdema due to circulatory disease, or nephritis, and as a result of new growths in the vicinity of the vessels of the neck, and finally as a variety of **angio-neurotic œdema.** This latter occurs in various parts of the body, either in the form of large urticarial wheals of the skin or mucous membrane, or as œdematous swelling of extensive skin areas. Sometimes the cause is not apparent, but sometimes it follows indulgence in certain food, just as in the case of urticaria. In a patient of mine it always followed the use of white wine. Occasionally a hereditary predisposition exists. Its localization in the larynx is not by any means a very rare event, but its danger is diminished by its usually short duration. In spite of this, however, it is quite conceivable that it would be fatal, if the obstruction of the glottis were complete. Indeed, death has resulted in such a case because medical assistance was not immediately available. But sometimes the œdema disappears as rapidly as it comes, even in the very presence of all the preparations made for tracheotomy. We must probably include in this category those cases of laryngeal œdema which have been observed in persons with an idiosyncrasy towards *iodide of potassium*. Asphyxia has even been observed in such cases.

The diagnosis is not difficult in cases of angio-neurotic œdema, because the patients are usually aware of the nature of their malady and are able to provide their own diagnosis quite accurately. It is quite different in the cases of idiosyncrasy towards iodide of potassium. In every case of unexplained laryngeal œdema, enquiries should be directed towards the administration of some form of iodine, if we have not ourselves prescribed it.

The diagnosis of laryngeal œdema is easy enough. It is quite possible to feel with the finger the two soft swellings which block up the entrance to the larynx, and a careful examination with the laryngoscope reveals an unmistakable picture.

(3) INJURIES.

Swellings of an inflammatory and circulatory character do not exhaust all the causes of acute dyspnœa. An external blow or stroke may lead to **fracture of a cartilage,** whether it be ossified or not, and the submucous hæmatoma resulting therefrom is liable to obstruct the glottis in a very short time. The presence of unnatural movement on careful external palpation, surgical emphysema, and the view of the hæmatoma with the laryngoscope, permit us to make the diagnosis. The most varied injuries of the respiratory tract may cause asphyxia by means of *surgical emphysema.*

(4) FOREIGN BODIES IN THE AIR PASSAGES.

The practitioner often sees another cause for sudden dyspnœa, in the inhalation of **foreign bodies.** We can realize the things which have been found in the air passages, if we think of what children put in their mouths and of what adults hold between their lips for convenience. Beans, peas, glass beads, bits of bone are of the most frequent occurrence ; nails, needles, and shirt buttons testify to the bad habit of using the lips as prehensile organs. Even pieces of dentures have found their way between the vocal cords, an incident which should always suggest unconscious inhalation in epileptic attacks.

If the circumstances point to the possibility of a foreign body having been inhaled, we must first find out whether it is really in the air passages. If it is acknowledged that it was put into the mouth, and this was followed by a severe attack of coughing and the vomiting of blood-stained phlegm, the probability is that the foreign body *was* in the air passages, but it may have been expelled by the coughing. The patient may still feel it there for a very long time, varying with his sensitiveness and the amount of injury inflicted on the mucous membrane—at any rate for a few hours. If there is no dyspnœa, there is ample time to use the laryngoscope. A negative finding, the disappearance of the cough without any artificial aid, and the absence of any lung symptoms suffice to reassure us. A foreign body situated more deeply, *i.e.*, in the trachea, manifests itself by attacks of coughing which are incited either on the under surface of the vocal cords or at the bifurcation of the trachea. A foreign body in the bronchus will assert itself by pulmonary symptoms on the corresponding side.

The accuracy of this last statement should be qualified. A foreign body may remain in the bronchus for a day or two without causing any symptoms. Thus I saw a youth who confessed to having swallowed a lead-pencil case, but who distinctly denied having any respiratory inconvenience at the moment of swallowing it—this he did in order to put his fault in as favourable a light as possible. It turned out afterwards that, as a matter of fact, he had a severe attack of suffocation. After the foreign body had entered the bronchus, there were no more subjective symptoms, but two days later the pneumonia which supervened, and the skiagram, showed that the metal case was in the left main bronchus. It was successfully removed with the foreign body forceps, through a tracheotomy incision.

If there is a persistent or paroxysmal cough, without any sign in the larynx, we must listen for a fluttering sound in the trachea. If this should be heard, it signifies that the foreign body is being wafted between the larynx and the bifurcation, with each breath, and that the cough is being excited from both positions. Such a case will be further elucidated by tracheotomy, when the offending substance may fly out with the first cough. If this does not happen we must look for it with the laryngoscope introduced into the trachea.

If the foreign body cannot be seen in the larynx, but is of such a nature as permits of its demonstration by X-rays, this measure must not be neglected (fig. 82). If this yields no result and the coughing still persists, we must either undertake a tracheotomy, with some misgiving, or refer the case to an expert in tracheoscopy and bronchoscopy.

The procedure must be quite different, if there is definite *dyspnœa* from the start. All diagnostic speculations must be abandoned and the urgent demand of the moment must be satisfied. When the patient can again breathe and our diagnosis is still unformed, the Röntgen rays and bronchoscopy may be invoked.

We have hitherto assumed that the dyspnœa is caused by the foreign body being situated in the air passages; but this is not always the case. I once had an epileptic on the operation table, who suffocated himself through swallowing his tooth-plate. It pressed from behind on the trachea, and I had to do tracheotomy before there was time to open the œsophagus to remove the plate.

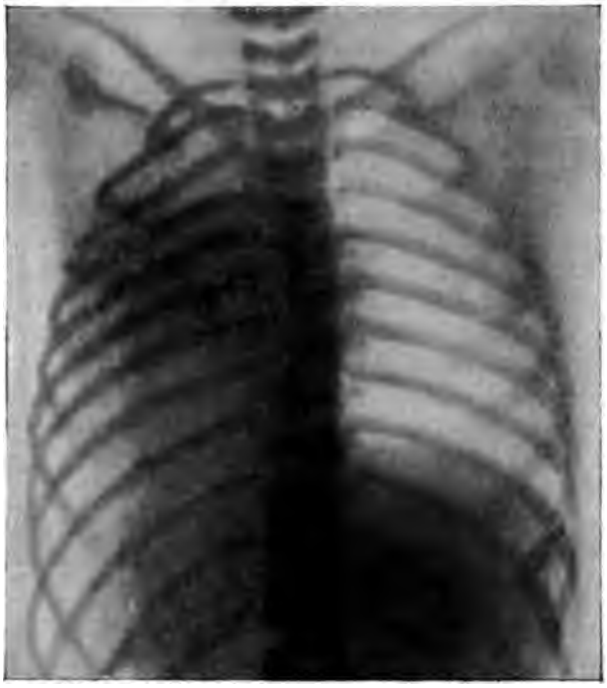


Fig. 82.—Metal case in end of left main bronchus. Lung tissue extensively thickened (pneumonia).

B.—CHRONIC DISEASES.

The problems are quite different if the laryngeal symptoms have come on *gradually*, in which circumstance the *previous history* is of the greatest importance.

A remark of a non-surgical character is worth making here. If a young or middle-aged person, who is neither a drinker, a heavy smoker, nor a speaker, and is otherwise healthy, becomes persistently hoarse, the possibility of syphilis should be thought of, especially the catarrhal hoarseness of the secondary stage.

If the trouble has begun with hoarseness, and there has developed, in the course of months, a persistent or paroxysmal dyspnœa, with difficulty in swallowing, *a tumour or ulcer of the larynx* must be suspected. We must, of course, be sure that we are not mistaking a tumour of the neck, such as a malignant growth of the thyroid or adjacent region, or a retropharyngeal tubercular abscess, for a laryngeal disease. A careful examination is necessary for this purpose. A small cancer of the thyroid may cause hoarseness by paralysing the recurrent laryngeal nerve and its pressure may cause both dyspnœa and difficulty in swallowing. This last differs in character from the same symptoms as caused by laryngeal ulcers, because the difficulty of swallowing is due to a *mechanical obstacle*, whereas in laryngeal disease the *pain of the act of swallowing* is the real subject of complaint.

The true nature of the disease can often be suspected before resorting to the laryngoscope. We need not hesitate about diagnosis if a typically tubercular looking patient states that he has been hoarse for years, brings up blood and suffers from night sweats. Of course a consumptive may have syphilis or cancer, but the long duration of the symptoms excludes the latter, and the syphilitic factor can be cleared up by means of Wassermann's test and specific treatment. It takes a *year* or more for a tubercular ulcer to effect the damage which cancer can do in a few *months* or a gummatous ulcer in a few *weeks*. The most significant point in the history of *tubercle* is the early onset of pain on swallowing, which is occasionally much more pronounced than difficulty in breathing.

Too much deference must not be paid to the previous history, to the neglect of careful examination. The following case will convey this moral.

A remarkably healthy young woman, hardly 30 years of age, sought advice about difficulty in swallowing, which had persisted for some months, but which had lately become worse. Her main symptoms were pain and a feeling of soreness. The laryngoscope showed an ulcer between the arytenoid cartilages, spreading towards the back of the larynx. Syphilis could be excluded, and the case appeared to be clinically tubercle, but the microscopical examination of a small piece of the margin of the ulcer declared it to be carcinoma. Before the patient agreed to an operation she was suddenly attacked by aspiration pneumonia. In this case the position of the ulcer was certainly unusual for tubercle, and its situation in the inter-arytenoid region towards the back of the larynx might have suggested the possibility of carcinoma. Since then I have seen a similar case, also in a young female patient. It should be added that Sendziak's statistics show that cancer in this particular situation is much more frequent in women than in men.

If a healthy man of middle or advanced age begins to get hoarse, and if this hoarseness, after some months, is accompanied by some slight difficulty in breathing, we should think, instinctively, of cancer and examine the neck for hard, enlarged glands. The absence of such glands is no argument *against*, but their presence is a strong evidence *for* it, provided of course that this glandular enlargement is recent. A pronounced fætor *ex ore* is also in favour of cancer, because this symptom is very rare in other ulcers. Some support to the diagnosis is afforded by the knowledge that the patient is a lover of the bottle. Usually tobacco is blamed quite confidently; people prefer to be known as inveterate smokers rather than as habitual drinkers.

Age plays so important a rôle in regard to cancer of the larynx, that *chronic hoarseness coming on in a man over* 50 *must be suspected to be due to cancer.*

But at the same time we must discard all preconceived ideas, as the following case shows.

An old man was sent to the surgeon with the diagnosis of cancer, because he was suffering from hoarseness and difficulty in swallowing. The surgeon refused to operate, and for a good reason, because despite a late appearance, the patient had pronounced pulmonary tuberculosis, and his ulcer was not cancerous but tubercular.

If the patient states at once that he has had syphilis, we must not forthwith conclude that he has a **gummatous ulcer,** but we should, at any rate, institute specific treatment. The propriety of this course would be confirmed if the patient is neither tubercular, nor at the cancer age, if glandular enlargement is absent and if the subjective complaints are slight, and above all if other traces of tertiary syphilis can be detected.

The history and the general condition having put us on the track, we must next proceed to an *examination with the laryngoscope.* In the first place, this informs us whether the hoarseness is the result of paralysis of the recurrent laryngeal nerve (aneurism, goitre, mediastinal tumour, apical tuberculosis). If any anatomical change is discovered in the larynx the *situation* of the lesion is full of suggestion.

Tubercle prefers the vocal cords, the neighbourhood of the arytenoids, and less frequently the epiglottis. *Syphilis* rather prefers the latter situation, but may occur anywhere in the larynx. *Cancer* is most frequently found on the vocal cords and then in decreasing frequency on the ventricular bands, the epiglottis, and the posterior wall of the larynx.

The greatest care is required in basing a diagnosis on the *clinical features* of the disease. All the three forms may start as nodules, and all three subsequently ulcerate. If there are a *number of nodules* present, we should think of *tubercle* or *gumma* rather than of *cancer.* A cauliflower-like shaggy appearance indicates cancer. That it is not easy for the inexperienced to decide from the appearance of the ulcer is obvious enough if we consider the difficulty which even the experienced find in diagnosing an ulcer of the oral mucous membrane, quite accessible to the eye. The rules we have already mentioned in connection with the latter ulcers also apply in this instance. Ulcers and growths which extend beyond the boundaries of the larynx, encroaching upon neighbouring organs, are most likely cancerous. In such cases, evidence of a hard margin and base, which can easily be obtained by palpation, is often of decisive significance.

Often enough the laryngoscope does not permit us to get beyond the mere diagnosis of "ulcer." Three other methods may then be resorted to: histological and bacteriological examination and therapeutic experiment.

The piece submitted to *histological examination* should not be too small. It may be removed by the practitioner if he possesses sufficient dexterity, otherwise this procedure should be carried out by the laryngologist. It is most important that the piece should be snipped off the right place. An error in this respect may be disastrous in its consequences, as testified by a famous historical instance which it is unnecessary to recall.

There is no difficulty in recognizing *cancer* under the microscope. The distinction between *tubercle* and *syphilis* may be rather difficult if there is no pronounced general tuberculosis present, but here the gap will be supplied by *bacteriological examination*. It may be possible to detect tubercle bacilli by rubbing the surface of the ulcer with a laryngeal probe covered with cotton wool, and wiping the wool on a cover glass. Animal inoculation with a portion of test specimen removed is, however, a much more certain method. The experiment of treatment with *iodide of potassium* must be regarded as an ultimate resort, if Wassermann's reaction is positive.

It happens sometimes that although the symptoms have led us to suspect tubercle, syphilis, or cancer, the laryngoscope reveals a sharply circumscribed structure, situated on a vocal cord or on the anterior commissure, with absolutely no morbid change round about. This is, in all probability, an *innocent growth*, and if it is smooth on its surface like a pea or somewhat rough from being an aggregation of separate roundish little nodules, it may be regarded as a **fibroma.** If the growth looks like a cauliflower or a condyloma with points, it is to be regarded as a **papilloma.** Such papillomata do not always exist in the form of circumscribed tumours; they may extend superficially just like papillomata of the bladder.

Can these innocent tumours not be diagnosed *clinically?* In some cases, certainly they can; in cases wherein the growth is pedunculated and gets caught occasionally between the cords. The history will show that the patient sometimes has a clear voice, and then suddenly gets attacked by hoarseness or even seized by suffocation. If a child suffers from unexplained persistent hoarseness or from repeated attacks of unexplained suffocation, we should think of a papilloma, because this is not at all a rare condition in children, and is practically the sole laryngeal tumour which occurs among them.

Errors in diagnosis in respect to innocent laryngeal tumours may be made in various directions. A circumscribed tubercular or gummatous nodule may be mistaken for a fibroma or *vice versa*, but the further course of the case would, however, clear this up. In other cases we may be doubtful whether a papillary structure which we have discovered is innocent or malignant. Age is, of course, of great significance here, for a papilloma in an old man is always suspicious of cancer, and if enlarged hard glands are present, the matter is conclusive. One must never wait for enlarged glands to confirm the diagnosis before operating, because in cancer of the larynx the glands are often very late in appearing. When in doubt the only course to pursue is to have an adequate portion excised for examination.

There are some rare laryngeal tumours which cannot usually be diagnosed until after their removal, such as the peculiar amyloid tumours. The practitioner cannot be expected to recognize these accurately. The same applies to tumours of the *trachea*. It may be mentioned as a curiosity that *new growths with the structure of the thyroid gland* have been found in it, obviously arising from a misplaced thyroid rudiment. *Sarcoma* occurs more frequently, and therefore has more practical importance. The diagnosis of a tumour of the trachea is made by a process of exclusion, if no other explanation for the difficulty in breathing is forthcoming. An expert in the use of the laryngoscope may be able to see the tumour even in this situation. If unsuccessful at first the patient must give several sittings in order to become gradually accustomed to the examination, as is so often necessary when laryngoscopy is not well tolerated.

It must be mentioned that tumours, especially cancer, in the vicinity, may infiltrate the trachea, and grow in a fungiform manner. As a rule by the time a primary cancer has manifested symptoms pointing to the trachea, it has already declared itself in other ways, so that the diagnosis is attended by no difficulty.

CHAPTER XXI.

DIFFICULTY IN SWALLOWING.

In accordance with an old and useful rule, we must distinguish the difficulty in swallowing caused by some disturbance of the mechanism in the mouth or throat, from the difficulty caused by obstruction in the œsophagus. Obviously this difference implies the existence of very different conditions.

A.—DISTURBANCES OF THE MECHANISM OF SWALLOWING IN THE MOUTH AND THROAT.

Deglutition may be deranged in various ways :—

(1) *Paralysis of the muscles of the palate.* In order to swallow efficiently it is necessary that the upper portion of the pharynx should be shut off by the action of the soft palate. If the latter is paralysed it follows that some of the food will gain access to the nose by escaping upwards. This upward flow will not affect the food alone, it will also affect the current of air during speech, and therefore the nasal intonation of the patient will suggest the cause of the difficulty in swallowing, before even we make an examination. *Paralysis of the palate after diphtheria* is a classical example of this condition. The paralytic symptoms in *bulbar palsy* are much more extensive, but in this disease the difficulty in deglutition is preceded by many other paralytic symptoms which will already have established the diagnosis.

(2) *Congenital or inherited defects in the development of the soft palate* may interfere with the act of deglutition, just like paralysis. The former condition is usually associated with a *cleft of the hard palate,* and the latter is the result of *gummatous* destruction, but a patient with cleft palate is more or less able to compensate for his disability by raising his tongue to close the fissure.

(3) *Scars,* especially after tertiary syphilis, more rarely after burns and corrosions, may interfere with the mobility of the palate and thus prevent the effectual shutting of the pharynx.

(4) *Pain* is a frequent cause of difficulty in swallowing, for when severe it may completely inhibit deglutition by reflex action. Every layman recognizes the difficulty in swallowing in cases of sore throat; but it is also a special complication of laryngeal tuberculosis, as also of laryngeal and pharyngeal cancer. In tubercle, the difficulty may be so great that feeding becomes almost impossible. The difficulty in swallowing when a foreign body is in the throat, especially in the pyriform fossa, is also due to the inhibition caused by the pain.

(5) Apart from the inhibition caused by pain as just mentioned,

acute inflammatory processes interfere with the act of deglutition, and may even render it impossible. This is due to inflammatory infiltration of the soft palate, to diffuse swelling of the entire throat, and to extreme bulging of the affected side, if an abscess has formed. The swelling in phlegmonous inflammation of the floor of the mouth and of the tongue causes a similar disturbance.

(6) Swallowing may be mechanically prevented by *pharyngeal tumours* of various kinds, such as *naso-pharyngeal polypi, naso-pharyngeal fibromata, retro-pharyngeal tumours* and *malignant growths* in any part of the throat. A chronic retro-pharyngeal abscess, generally tubercular, acts in the same way.

(7) The presence of a *foreign body* in the upper part of the pharynx also acts as a mechanical obstacle to deglutition.

The circumstances attendant upon the case will put some limit on the above mentioned possibilities, even before we examine the patient's throat.

A *sudden onset* of difficulty in swallowing, in a healthy person, indicates a **foreign body,** for which search must at once be made, either with the laryngoscope or the finger. If a foreign body is present, it is probably situated in the sinus pyriformis, or behind the larynx above the cricoid cartilage. I have seen a slice of radish sticking there on one occasion, and on another two pieces of tough unmasticated tongue.

If the trouble has come on *gradually*, or at any rate has not come on within an hour or two, the *age* must be taken into consideration first of all. Palatal paralysis in a child will suggest a sequela of diphtheria; in an adult it will point to bulbar palsy. If the difficulty seems to be due to a tumour, a retro-pharyngeal abscess is the most likely cause in a child, in a young person a naso-pharyngeal fibroma is probably responsible, and after 50 the chances are in favour of a malignant neoplasm.

The *voice* is very characteristic. A nasal tone means that the palate does not close up the pharynx satisfactorily, owing either to paralysis or structural defect. If the voice is hoarse, we think instinctively of laryngeal tuberculosis in young people, and of carcinoma in those of a more advanced age. But first impressions must not be permitted to lead us astray, they are only of value as initial guides, as the two cases previously noted will exemplify (pp. 126, 127).

Having made a provisional diagnosis from the history and the external circumstances, we next proceed to an *examination of the mouth and throat.* Often enough, one glance into the open mouth suffices for a diagnosis. A lax, dependent soft palate, remaining so even on phonation, indicates paralysis. Sore throat and retro-tonsillar abscess are obvious at once. In retro-pharyngeal abscess the posterior wall of the throat bulges forward. If nothing abnormal is seen, we must palpate the post-nasal space, and finally bring the laryngoscope to our aid. If nothing still appears, we must conclude that the trouble is not in the throat but in the region of the œsophagus.

B.—DISTURBANCES OF THE MECHANISM OF DEGLUTITION IN THE REGION OF THE ŒSOPHAGUS.

We now come to the second and most important group of deglutition troubles, to those which depend upon diseases of the *œsophagus and its vicinity*.

(1) If the *difficulty has occurred suddenly*, we must first think of a **foreign body** in the throat. In a large number of cases it will be found that artificial teeth, pieces of bone or coins, have been swallowed. But clear as the history is in some cases, in others we can elicit nothing of value. An epileptic who misses his artificial plate after an attack will look for it everywhere except in his œsophagus, as long as he feels no discomfort. I have myself seen a case wherein an agricultural labourer swallowed a piece of a goat's skull with his soup, and at first paid no heed to the incident. A child who has swallowed a coin, even if able to speak, will only vouchsafe information about his misdeed when he finds himself in difficulties. So that even if there be no history available, the sudden onset of difficulty in swallowing should always arouse first the suspicion of a foreign body. But how can the diagnosis be established? If the epileptic just referred to consults us a couple of days after he has missed his plate, because he is quite unable to swallow, and some inflammation is already beginning, we are forthwith in a position to tell him the whereabouts of the lost object. But in the absence of such clear indications, we ask the patient to take a little drink carefully, and point out the site of any pain which may be present. But some caution is required in this matter, because the localization of pain is limited to the upper portion of the œsophagus, and even if pain is ascribed to some definite spot in the throat, this only suggests that some injury has occurred there, and not that the foreign body is to be found there. We next proceed to pass a sound, beginning with a soft india-rubber one for the sake of gentleness. If this should be held up in its passage downwards, it will afford approximate information as to the site of the obstruction. A very flexible whalebone sound gives more reliable information. If it is conjectured that the foreign body has been swallowed a day or two previously, or even before that, the most extreme care will be required, lest inflammatory change has already supervened. In any case we should begin with a

sponge sound, after assuring ourselves that the sponge has been carefully cleansed and fits in firmly.

Accidents may happen even to the most expert in the passing of œsophageal bougies. Kocher had a case wherein he had to remove a piece of whalebone sound by gastrotomy. It was the first operation of the kind, deliberately carried out on a stomach not secured by adhesions.

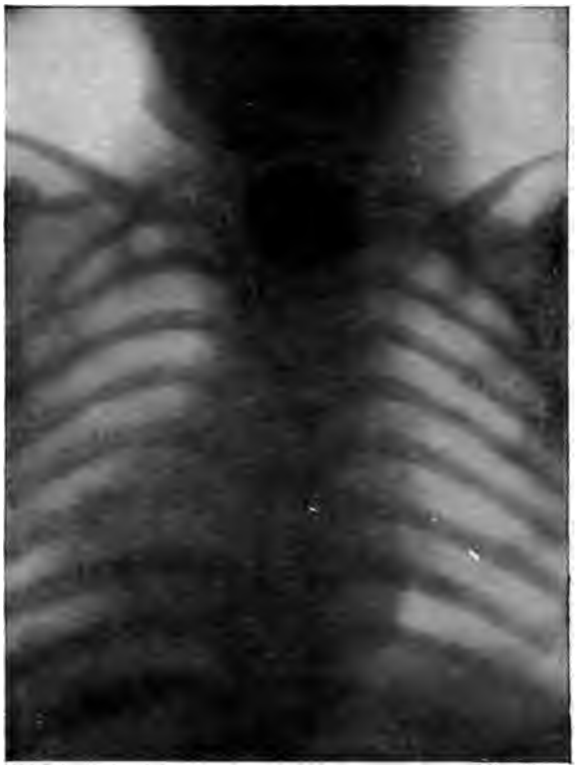


FIG. 83.—Copper coin in the œsophagus (for three weeks).

If we come to an obstruction on carefully pushing the sponge sound forwards, we must not proceed further, but withdraw it, and see whether there be any blood or pus on it. It will sometimes be necessary to wipe the sponge on a cover-glass, and stain with methylene blue, in order to detect the pus. We may next use a firmer sound, which will permit us to estimate the distance of the obstruction from the upper teeth, better than is possible with a soft sound. If we do not know the nature of the foreign body, we should employ a whalebone sound with a metal or ivory top, in order to determine its nature by the sensation experienced when the sound impinges upon it. But if the sound encounters no obstruction, we cannot exclude a foreign body, for even an artificial plate may be passed by the instrument without detecting it.

In all cases, but especially in doubtful ones, Röntgen rays should be employed, or a skiagram taken. Coins appear most distinctly (fig. 83), but in a successful picture, pieces of bone, teeth, and the metal portion of dentures may be visible. As a last resource, the expert may proceed to use the œsophagoscope, which will provide the truest solution.

Matters are, however, not concluded with the diagnosis of a foreign body. Treatment and prognosis demand some information as to the presence of an injury to the œsophagus, or of a pressure ulcer with an incipient peri-œsophageal phlegmon. In these circumstances the attempts at extraction must be most carefully conducted, the prognosis must be guarded, and the patient must be attentively watched, even after the successful removal of the foreign body. In order that we should not be blamed for symptoms which already exist, we must take the temperature and pulse *before* the extraction; and if the foreign body is in the upper portion of the œsophagus, we must note whether severe pain on pressure, swelling and œdema in the neck, point to the existence of a peri-œsophageal phlegmon.

It seems incredible that a foreign body may lodge in the œsophagus for quite a long time without causing severe symptoms. In the case illustrated in fig. 83, the copper coin had been in the œsophagus for three weeks, and there was such an absence of symptoms that both the doctor and the mother doubted its presence before the skiagram was taken. On the other hand, a youth swallowed a button from a uniform, and died a few days later, owing to the development of phlegmonous peri-œsophagitis.

Besides foreign bodies, the causes for acute onset of difficulty in deglutition include *corrosion by alkalis or acids, compression by an acute thyroiditis, acute goitrous inflammation*, or a *phlegmon of the neck or mediastinum.*

An accurate history is usually available in cases of **corrosion,** except in children, hysterical individuals, and pronounced mental patients. The very severe initial symptoms generally abate, but in the course of three or four weeks they are replaced by a gradually increasing stricture. In these cases great significance attaches to the persistent regurgitation or vomiting of blood-stained fluid.

Inflammatory processes in the neck are at once recognized by appearance and by palpation, and a **mediastinal phlegmon,** independent of a foreign body or cancerous disease of the gullet or bronchi, is a very great rarity.

(2) If the difficulty in swallowing has come on *gradually*, the cause

may be a **narrowing of the œsophagus,** or **external pressure** thereon, or a **functional disturbance.** The first occurs in cancer and cicatricial stricture after ulcers—mostly due to corrosion or syphilis ; and external pressure results from tumours of the neck, changes in the shape of the spinal column, aneurisms, mediastinal tumours of any kind, cold abscesses, and diverticula of the œsophagus.

Before we proceed to use the sound, there are other matters to note, because the instrument is not always safe, and because not all patients are equally agreeable to its employment. We must, above all, be able to exclude *aneurism.* For this purpose, it is necessary to begin by the percussion and auscultation of the thoracic organs, and by examining for other signs of aneurism. If they are present, we should take care not to pass a sound. As there are some aneurisms which cannot be detected by the usual methods of diagnosis, it is therefore necessary to employ skiagraphy, or at any rate transillumination of the thorax.

It is necessary to note the *precise characteristics of the difficulty in swallowing,* because they point sometimes to the nature and situation of the obstruction. If the patient complains, in the early stage of his disease, of a constant expectoration of saliva, we must conclude that the obstruction is situated high up, so that the gullet cannot dilate above it. We must come to the same conclusion if a constant desire to swallow is the first symptom. But if the patient states that as soon as he takes a cup of tea or milk he forthwith vomits it, but that the vomit is not sour, that he frequently vomits insipid mucoid material between meals, we may suspect the case is not one of genuine vomiting, but merely of the evacuation of the spindle-shaped œsophageal dilatation which always forms above a deep-seated obstruction. This profuse regurgitation shows that the obstruction is deeply situated and that it is of some considerable duration. These cases have often been treated for months as gastric disorders. If we ascertain that the act of swallowing has gradually become difficult, that at first solid food well masticated and mixed with water went down, but that latterly only liquids can be taken ; if there is neither the constant desire to swallow, nor the profuse regurgitation of liquids just drunk, we should think of a gradually developing stenosis in the middle of the œsophagus. A glass of water will afford the opportunity of a test. If the patient begins to cough after the first or second draught, the obstruction is high up. But if he can quickly drink a half or the whole of the glass before it returns, the obstruction must be near the cardiac end ; the more water that can be swallowed the lower it is. The neck of the patient must be inspected during this procedure. If hard glands exist above the clavicle, and the patient complains of pains in the shoulder and back of the neck, there can be no doubt about the diagnosis of *cancer*. If one side of the neck fills up somewhat during the swallowing, we should think of a *diverticulum*.

Having proceded thus far with our examination we may now resort to the *sound*, provided that we have excluded an aneurism. By means of the sound we can distinguish between a true stricture and a stenosis produced by compression, just as we can distinguish a stricture of the urethra from hypertrophy of the prostate. If, despite difficulty in swallowing, a medium-sized or larger sound immediately passes into the stomach at each attempt, the case is one of a **tumour compressing the gullet from without.** If the sound is at one time arrested at a short distance, whereas at another time it passes easily, there can be no question about the diagnosis of a **diverticulum,** provided that the instrument has not been held up by a fungating projecting carcinoma which has not yet led to stricture. The presence of this last condition would be recognized by slight bleeding. If the neck becomes inflated on swallowing and this swollen part can be emptied on pressure, the diagnosis of diverticulum is confirmed, and it is superfluous to render the stricture visible by filling with bismuth and applying the Röntgen rays.

The question whether a case is to be grouped under *pressure* or *traction diverticula* is easily settled. The former alone, when full, cause blocking of the upper part of the gullet, and produce clinical symptoms. They are usually situated in the cervical portion of the œsophagus and only exceptionally in the thoracic portion. The latter, when they are large enough, cause the usual symptoms of diverticula, even swelling up to the neck. Absolute accuracy of diagnosis can only be assured by Röntgen-rays. Traction diverticula are usually discovered *post mortem*, and the sound has no special tendency to catch in them. They hardly enter at all into clinical consideration. Traction diverticula seldom dilate through pressure.

Spindle-shaped dilatation of the œsophagus, previously noted and dependent upon some functional disturbance, must not be confused with a diverticulum.

It is certain that this condition is usually caused by spasm of the cardiac end of stomach ; but the possibility of it being caused in consequence of paralysis of the œsophageal muscle cannot be excluded. Solid food may remain in the œsophagus for days at a time. The sound can be moved about in the œsophagus remarkably easily, and it occasionally catches in its wall, so that a diverticulum would suggest itself, if it were not so deeply situated. The emaciation is often very pronounced, but even after many years' duration, it does not reach the extreme degree which is usually present after one year's suffering from cancer of the œsophagus. As in the case of a diverticulum, the presence of this dilatation can be demonstrated by the Röntgen-rays after a bismuth meal.

In severe cases the deviation of the œsophagus to the left is distinctive. The edge of the œsophageal shadow is invariably sharply defined, which is not always the case with cancer.

If the œsophagus is not permeable to a soft sound of medium thickness (10 mm.), we should endeavour to pass an olivary sound, beginning with one of a somewhat smaller calibre, but not the smallest, because this is much more liable to penetrate a putrefying growth than a thicker one.

Having overcome the first difficulty, the cricoid cartilage at a distance of 15 cm. (6 inches) from the upper incisors, the sound passes along easily until the stenosis is encountered. If delay arises here, the sound is withdrawn somewhat and then pushed on again, in case it may have caught against some projection of the new growth. But if this manœuvre does not succeed in passing the sound further on, without violence, we must try smaller sounds until we hit upon the one which just goes through. The sound is pushed right on into the stomach to see whether there is only *one* obstruction, and in withdrawing the instrument we note the exact spot where it is grasped so as to determine the *lower* limit of the obstruction.

This point is sometimes of practical importance, because carcinomata, whose lower limit is not more than 20 cm. (8 in.) from the incisors, can be removed through the neck, whereas more deeply placed carcinomata are attacked by modern surgery through the thoracic or abdominal route—unfortunately with very meagre success hitherto.

It is obvious that a physiological obstruction must not be mistaken for a pathological one; the cricoid cartilage, situated 15 cm. from the upper incisors, has already been mentioned. A little resilience is also felt at the level of the bifurcation of the trachea (26-67 cm.), and this is a little more pronounced when the sound passes through the œsophageal opening in the diaphragm (38 cm.). We must not be deceived by spastic constriction which occurs now and again in nervous individuals, and which may prevent the passage of the sound, analogous to the case of urethral spasm. We should conclude that this kind of difficulty is present if the condition varies from time to time while genuine signs of diverticula are absent.

If we have found a *stricture*, we must next decide whether it is cancerous or cicatricial. A patient never forgets having drunk a corrosive acid or alkali, and therefore the evidence for a stricture from **corrosion** is at once available; so that in practice the diagnostic distinction usually concerns **cancerous and syphilitic stricture.** A recently developed hard gland in the supraclavicular region points to cancer.

There is no connection between the size and age of the cancer and the extent of the glandular enlargement. I have seen a bunch of glands as large as a fist, when there was clinically nothing at all definite, the autopsy only revealing a growth of 2 cm., which had not formed a ring, but allowed even the largest sound to pass. On the other hand, it is often impossible to feel any enlargement of the supraclavicular glands, although the cancer be of great extent.

Age, addiction to tobacco or alcohol only afford approximate indications. But a history of old syphilis, and especially the presence of other tertiary signs, is of great significance when enlarged glands are absent.

The course of the disease is conclusive from the clinical standpoint. A syphilitic stricture may develop more quickly than cancer, but once having formed, it does not increase continuously like a cancerous stricture. Neither is syphilitic stricture attended by the spontaneous pain which is seldom absent in advancing cancer.

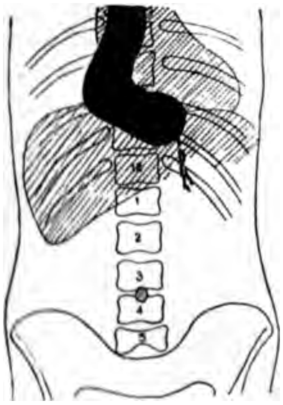


FIG. 84*a*.—Cardio-spasm. The limits of the shadow are well defined.

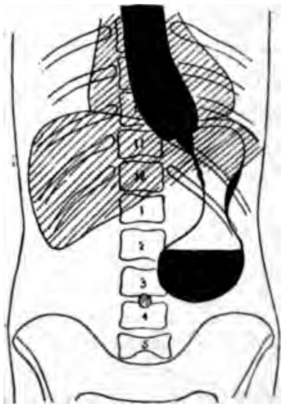


FIG. 84*b*.—Cancer of œsophagus. Conical process of the test mixture.

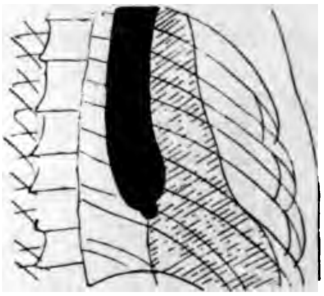


FIG. 84*c*.—Cancer of œsophagus. Taken diagonally from right anterior to left posterior. Conical process of shadow.

The diagnosis may be confirmed by œsophagoscopy, by aid of which a piece of the tissue in question can be removed for examination. But cancerous stricture is so much more frequent than syphilitic stricture, that we may well dispense with this method. *A man of advancing years, suffering from a gradually increasing stricture of the œsophagus is in all probability a victim of carcinoma.* Œsophagoscopy is indispensable in those somewhat rare cases of indefinite and intermittent dysphagia, which are suggestive of new growth, but yet which admit the passage of a large-sized bougie quite easily. Direct inspection may enable an early diagnosis to be made in this type of case.

A skiagram is most valuable in deep-seated obstruction, because it forthwith establishes the differential diagnosis between carcinoma and a deep-seated diverticulum, and often reveals the existence of cardio-spasm. The border of the shadow is often irregular in cancer, and frequently exhibits a conical process. In cardiospasm the shadow may be circular or conical, but its edge is always sharply defined (fig. 84, *a*, *b*, *c*).

In addition to syphilis and the action of corrosives, certain other rare causes of stricture have been noted, viz., non-traumatic ulcers, peptic ulcers of the lower part of the œsophagus and peri-œsophageal abscesses.

Sarcoma of the œsophagus is very rare, and can only be diagnosed with the microscope.

CHAPTER XXII.

ABSCESSES OF THE NECK.

THE questions involved in acute abscesses and acute phlegmons of the neck are so different from those which present themselves in chronic abscesses, that we separate the two classes, although intermediate forms do occur.

A.—ACUTE INFLAMMATORY PROCESSES.

In cases of phlegmon or abscess of the neck, our first concern is to ascertain the position of the inflammation. If the latter corresponds to the known situation of glands, we shall not be wrong in assuming that there is *suppuration of the glands*, but our task is not completed therewith. Glands do not suppurate of themselves; there must be some antecedent infection from without, and the suppuration represents the attempt of the glands to prevent the micro-organisms gaining further access into the system. We must, therefore, search for the *portal of entry*. If the sub-mental or sub-maxillary glands or those along the large vessels are involved, there may be some easily visible skin infection such as a furuncle, but the origin is more probably in the mouth or pharynx, especially in connection with the gums, teeth, or tonsils. This kind of cervical abscess is most prevalent after scarlet fever and diphtheria. Sometimes the inflammation at the portal of entry is so slight that the most careful examination is required to detect it. If the glands at the back of the neck are suppurating, a condition to which children are prone, the experienced practitioner will at once look for an eczema of the scalp, and he will rarely be disappointed.

We will now discuss the various regions of the neck.

(1) THE SUB-MENTAL REGION.

Sub-mental abscesses are easy to recognize, for they almost always arise from the *lymphatic glands* and not merely from the jaw. The portal of entry for the infection is usually situated on the upper lip or chin. A persistent abscess in this neighbourhood, which fails to heal after incision, must be regarded as a *suppurating dermoid* of the floor of the mouth, although these usually grow towards the mouth. The sub-lingual gland very rarely comes into question, and it is so situated that if it does inflame the swelling is inwards.

In acute glossitis and in cellulitis of the floor of the mouth there is also œdematous swelling of the sub-mental region.

(2) THE SUB-MAXILLARY REGION.

Acute inflammatory swellings of the **sub-maxillary region** may be due to one of the following causes :—

(*a*) *Periostitis of the jaw* after caries of the teeth. Examination of the teeth and gums, palpation of the jaw will indicate the existence and situation of the inflammation.

(*b*) *Osteo-myelitis of the jaw.* This differs from periostitis by its great extent (usually bilateral) and the greater severity of the general symptoms.

(*c*) *Acute inflammation of the sub-maxillary gland* (*salivary*), through obstruction of Wharton's duct (salivary calculus), infection from the mouth, or as part of an epidemic parotitis. The gland can be more or less easily felt, both in the mouth and externally. Sometimes pus exudes from Wharton's duct when pressure is made on the tumour. The occurrence of repeated attacks suggests calculus, which can occasionally be felt within the mouth, and a skiagram can be obtained with a suitable arrangement of the plate.

(*d*) Inflammation of the lymphatic gland tissue enclosed in the capsule of the salivary glands, so called *angina ludovici.* Here also the swelling projects mainly into the mouth, but the general symptoms of infection are much more severe than in inflammation of the salivary glands, and there is in addition a very widespread œdema.

(*e*) Inflammation of the *superficial lymphatic glands* superjacent to the salivary glands. This is the most frequent form of sub-maxillary phlegmon, and chiefly spreads outwards. It causes fluctuation much more quickly than intra-capsular suppuration, and is less dangerous. The infection may start from the nose, eye, cheek or gums.

A *laryngeal perichondritis* may open externally in this direction, and suppurative *periostitis of the hyoid bone* may be mentioned as a curiosity.

(3) THE SIDE OF THE NECK.

Practically the only condition which presents itself at the **side of the neck,** in the sterno-mastoid region, is *glandular abscess.* If no focus of infection is found on the skin or mucous membrane we must assume that the original disease, eczema of the head, rhagades on the nose, and inflammation of the gums, &c., had already recovered when the abscess came on. Injury to the mucous membrane of the pharynx or gullet by a fish-bone or similar pointed foreign body is a rarer cause of cervical abscess. Abscesses tracking from the *œsophagus* always appear first in the neighbourhood of the sterno-mastoid, and are recognized by the fact that they cause pain on swallowing from the very beginning. The patient will also, as a rule, be able to recall the severe sudden pain caused by the foreign body responsible for the mischief. Pain on deep pressure can usually be elicited before any inflammatory change appears on the skin; and the abscess does not reach the surface in the form of a circumscribed swelling but as a diffuse phlegmon.

(4) SUPRA-CLAVICULAR REGION.

Abscesses and phlegmons in the **supra-clavicular fossa** are less frequent. The glands in this position very rarely suppurate, because the infective organisms are usually arrested by glands higher up. The abscesses which do occur in this region are usually due to extension of suppuration from above. If a phlegmon does arise primarily in the supra-clavicular fossa, we must think of *osteo-myelitis of the clavicle.*

(5) ANTERIOR TRIANGLE OF THE NECK.

We now proceed to the somewhat rare abscesses of the **anterior triangle of the neck.** They usually originate in the *thyroid gland,* whether the change therein is only goitrous or not. If the patient is seen in an early stage when the inflammatory process is limited to the thyroid gland or to a goitre, there is no difficulty in the diagnosis. But if the patient is not seen until a state of diffuse phlegmon has supervened, it may be necessary to consider the possibility of *myositis of the sterno-mastoid. Osteomyelitis of the manubrium sterni,* with the pus tracking upwards, might also be mistaken for inflammation of the thyroid gland; but the pain on pressure over the sternum should guide us to a correct diagnosis.

Finally, there are *phlegmons of the anterior mediastinum* which appear in the neck. I have seen two cases which ended fatally, despite early incision.

(6) POSTERIOR CERVICAL REGION.

Abscesses in the **posterior cervical** region and its neighbourhood are more frequent. If the abscess is situated *posteriorly, below the mastoid process*, and has been preceded by middle-ear symptoms, we cannot fail to diagnose a *Bezold's abscess, i.e.,* an abscess of the mastoid process which has burst in the neck.

How can this latter variety of abscess be distinguished from one of the ordinary superficial glandular abscesses of this region? An abscess starting from the petrous bone, or mastoid process, is at first deeply situated and covered by the insertion of the sterno-mastoid. The patient therefore complains of pain and holds his head stiff, before any swelling or redness of the skin appears. But, on the other hand, glandular abscesses are from the first quite close to the skin; the subjective disturbances, therefore, go hand in hand with the visible and palpable development of the abscess.

A carbuncle at the back of the neck presents a very distinctive picture. The simple posterior cervical furuncle demands no diagnostic skill; and it is not easy to mistake a carbuncle formed by the agglomeration of a group of contiguous furuncles—sometimes called anthrax, after its French designation.

Both terms occasionally mislead the beginner into thinking of true anthrax. Before the advent of bacteriology the differential diagnosis was often difficult. As a matter of fact the typical carbuncle at the back of the neck has nothing to do with anthrax. It is a staphylomycosis which finds a favourite soil for growth in the aged or diabetic.

The inflammatory process is not always limited to the diseased hair follicles and their immediate vicinity. The entire integument of the back of the neck, from ear to ear, may become indurated and of a bluish-red colour. The persistence of fever after the evacuation of cores of pus shows that some deep inflammation exists. A phlegmon may develop over a muscle, beneath superficial skin which is perforated like a sieve, but the cutaneous infiltration renders the detection of fluctuation very difficult. Nevertheless, it is important to open such a phlegmon as quickly as possible, in order to anticipate the deep extension of the pus.

If there is no furuncle to explain the condition, disease of the bone must be thought of, *i.e., osteo-myelitis of the occipital bone*, which is more likely to be metastatic than primary.

Suppuration of a tumour at the back of the neck is rarer still. While acting as assistant, I saw a girl with a large fistulous abscess at the back of the neck constantly discharging fœtid pus. It was a case of a *dermoid*, containing a large bunch of hair. *Lipomata* may also suppurate, exceptionally.

B.—CHRONIC ABSCESSES.

A slowly developing, painless and circumscribed swelling at the back of the neck should suggest a new growth, and this diagnosis should only be abandoned in favour of a **tubercular or cold abscess** if all the circumstances are not in accord with it. The possibility of cold abscess has occasionally been forgotten, and immediate operation has been undertaken in the expectation of finding a tumour or a cyst. This error is easily avoided if the glands are suppurating, because the multiplicity of the swellings will indicate, even to the beginner, that glandular disease is present. But it is quite otherwise when a deep-lying abscess starts from a *tubercular vertebra*. If it has not yet reached the surface, the presence of fluctuation cannot always be detected, and the original specific disease may not have manifested itself in any striking manner. There is, however, even in this early stage, a symptom very significant of tubercular abscess, and that is the situation of the swelling *behind* the thyroid and *behind* the carotid, to which further reference will be made in discussing tumours of the neck. A swelling which pushes the carotid artery forward must be a tumour of the vertebral column, or of the deep cervical muscles, or a cold abscess.

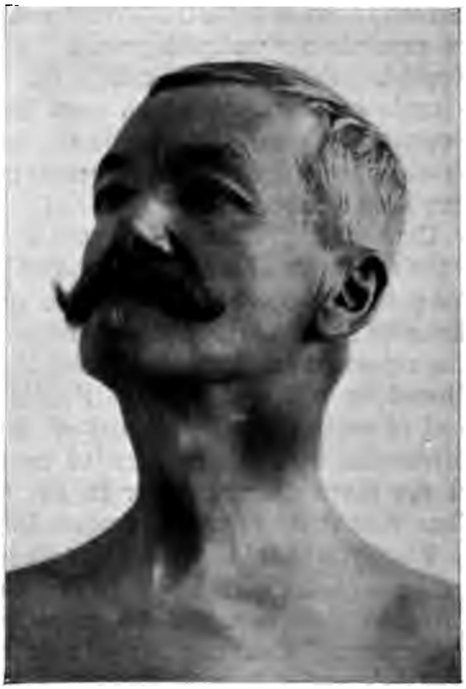


FIG. 85. — Tubercular abscess from spinal disease. Upper half of sterno-mastoid bulged forward.

If the pus has tracked to the surface by one route between the muscles, the deep origin of the abscess may be obscured. It is therefore always necessary to examine the inside of the throat, because the origin of the abscess may be easier to detect from there than from the surface of the neck.

A careful examination of the spine will generally reveal signs of tubercular disease before the patient is conscious of any trouble. The experienced eye will also note the stiffness of the neck, and will be struck by the fact that the patient never turns his head without turning his back at the same time.

A full *œsophageal diverticulum* projecting into the side of the neck may feel like a chronic abscess. But its long duration, extending over years, and its objective condition of expressibility render the diagnosis quite easy.

Not all chronic abscesses of the neck are tubercular. These are always distinguished by their softness, but there are some chronic abscesses in the neck which are, on the contrary, remarkably hard. Reclus applied to them the term "**phlegmon ligneux,**" and in German the expression "**holzphlegmone**" (board-like phlegmon) has been adopted. *Actinomycosis*, which we have already discussed, is one example of the cases which must be ascribed to this group. The occurrence of small soft areas in the midst of the board-like induration allows us to recognize actinomycosis before the characteristic granules have been discovered.

Cases of hard chronic inflammation, which are not to be attributed to the ray-fungus, have been found to be due to various schizomycetes. The patients are always in a feeble state of general health and of an advanced age. The board-like nature of a phelgmon does not, therefore, represent any specific disease; it is merely the distinctive reaction evinced by old cachectic individuals towards the presence of any kind of suppuration. Instead of the abscess breaking, it becomes surrounded by an induration of connective tissue, which renders it all the more difficult either to get absorbed or to burst through. Thus it is that the whole process is so protracted.

Finally, it should be noted that these "board-like phlegmons" must not be confused with Ludwig's angina, a mistake which has occurred, owing to their hardness on palpation. Notwithstanding the term "phlegmons," these cases are of a definitely chronic character, whereas Ludwig's angina is characterized by its acuteness. These cases have also often been mistaken for malignant growths, a matter to which we will refer subsequently.

CHAPTER XXIII.

SINUSES IN THE NECK.

SINUSES which result from *injuries* offer no diagnostic difficulties. The memory of the injury abides with the patient, and the direction of the sinus can be inferred by noting whether air bubbles through it, whether food issues from it or pure saliva flows therefrom. All other sinuses arise either from inflammatory processes (tubercle, syphilis, actinomycosis), or from *congenital anomalies.*

A correct diagnosis can usually be made from the *mode of origin, course,* and *external appearance.*

Gummatous inflammation does not lead to sinus formation, unless there be extensive destruction round about, and such a sinus does not persist very long, if no deeply situated organ has been perforated. The sinuses which are present in actinomycosis are situated, as previously mentioned, in hard board-like tissue; they are not drawn in, but are found on the summits of small dark-red projections of soft skin. Their duration is short, unless the disease has affected deeper organs, such as the spinal column and the base of the skull, when the sinuses may last for months, and become contracted while the characteristic changes of actinomycosis round about may become obliterated. In such cases there is always some secondary infection at work. **Tubercular** sinuses arise either from glands or from *tubercular foci in bone,* generally in the vertebral column. In the former case their duration is a matter of weeks, or at most months, and there will usually be present, among old scars, some remaining tubercular glands in various stages of suppurative softening. But sinuses arising from bone, such as in spinal caries, may persist for years. Here, however, signs of tubercular disease in the spinal column will always be present.

The points already mentioned will usually enable us to distinguish between a sinus due to *inflammation* and one due to *congenital causes.* The history will, as a rule, inform us whether the sinus dates from birth; but we must not exclude its congenital origin because the sinus first appeared in later years. It frequently happens that a branchial-cleft cyst persists in the deeper parts for years after birth, and then gradually makes its way to the surface, finally penetrating the skin and forming a sinus. Neither must we at once assume that a sinus has an inflammatory origin because signs of inflammation appear round about, for these branchial-cleft cysts are often the seat of inflammatory processes. Inflammation of a cyst alternates with the breaking out of the sinus, so that the patient complains of an "abscess in the neck," which empties itself from time to time, and which, after a longer or shorter existence, again develops a sinus. The inflammatory changes in cases of congenital sinus are of small extent, and abate as soon as the sinus develops. It appears as a small punctiform opening, surrounded by a somewhat in-drawn area of skin, either normal or slightly irritated by secretion. A sinus of many years' duration, with these characters, is almost certainly of congenital origin.

The *secretion* from a sinus affords us an additional diagnostic aid. Pure pus escapes from an inflammatory sinus; but in the case of actinomycosis it is occasionally mixed with the characteristic granules. Congenital sinuses discharge a purely mucoid or muco-purulent fluid, in which epithelial cells can be demonstrated as well as pus cells.

The *position* of the sinus also yields useful indications. Congenital sinuses are situated in the middle line, or in the region of the sterno-mastoid, whereas the sinuses of spinal disease, with which they may easily be confused, are generally located much more posteriorly. But if all this does not suffice, we may test the connection between the sinus and the oral or pharyngeal cavity by injecting some harmless colouring matter or a bitter fluid.

Finally, we may scrape some tissue off the wall of the sinus with a fine, sharp spoon. If, on examination, this is shown to be pure granulation tissue, with probably some tubercles, we are dealing with an inflammatory fistula; if epithelial cells are found, the case is one of congenital sinus.

Assuming that the diagnosis of **congenital sinus** is established, there still remain two questions to be answered.

(1) Is the case one of a *branchial-cleft sinus*, or is it one of so-called *median sinus*, arising from the thyreo-glossal duct?

(2) Is the sinus *complete* or *incomplete*, *i.e.*, is it connected with the throat or not?

The first question is answered by the position of the sinus. If it opens in the middle line, and it runs along the course of the middle line towards the hyoid bone, its origin in the thyreo-glossal duct is quite certain. If it opens at the side, it is usually a branchial-cleft sinus, arising from the first (very rarely the ear region), the second, or possibly from the third, branchial-cleft. Confusion may arise if a sinus from the thyreo-glossal duct opens laterally (fig. 86), and one from a branchial-cleft runs centrally, as sometimes happens. We must, therefore, note the course of the sinus as well as the point of its exit. The beginner may be inclined to use a probe in order to ascertain the direction of the sinus, but this is as dangerous as it is unreliable. It is very easy to penetrate the wall of the sinus with the end of a fine probe, and thus infect the surrounding tissue without ascertaining the direction of the sinus.

This must be determined mainly by palpation, for the tract of the sinus feels like a firm cord. As the course of the sinus which originates in the thyreo-glossal duct is a comparatively superficial one, we may be able to follow the whole tract as far as the body of the hyoid bone. If a sinus opens in the centre, and it is not possible to follow its course in this way, we must assume that the passage runs deeply, and *to the side*, that is to say, that it is the sinus of a branchial-cleft. If one has the opportunity for an X-ray examination, some bismuth emulsion should be injected into the sinus, and its course followed either on the screen or in the skiagram.

There are some cases wherein the microscope affords the first definite differentiation. Sinuses originating in the thyreo-glossal duct are generally surrounded by minute lobules of thyroid gland tissue, a feature which is, of course, absent in the cases of sinuses from genuine branchial clefts.

To determine whether the sinus is *complete*, and reaches as far as the throat, we must adopt the method of injection, as already observed. In the case of a sinus of the thyreo-glossa duct the coloured fluid will appear at the foramen cæcum, in front of the epiglottis; in the case of a branchial-cleft sinus it will appear at the lateral pharyngeal wall.

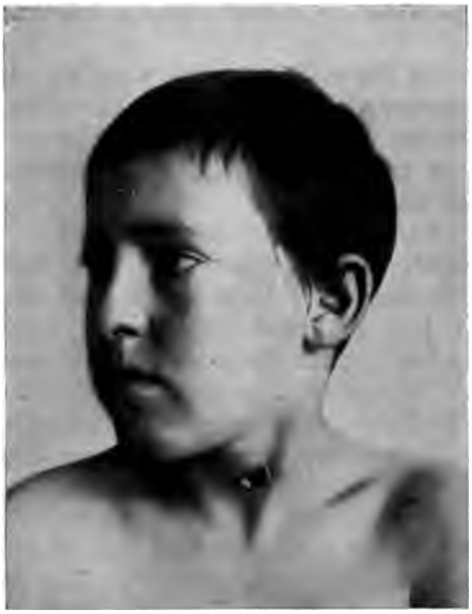


FIG. 86.—Congenital sinus of neck, originating in the middle but opening at the side.

Finally, there remains one special form of sinus, with its orifice at the front or side of the neck. It is usually situated in the scar of an incision, and thus shows its artificial origin. When the patient swallows, it rises upwards and forwards with the thyroid gland, and if a probe is carefully introduced, it loses itself in a swelling which can be identified as a goitre. The case is therefore one of a *sinus from a goitre*, originating after incision of a goitrous abscess. Such sinuses may persist for years, if the inflammation has developed in a cystic goitre with calcified walls. The sinus can only be radically cured either by removing the whole cyst or by extirpating all the tissue which is incapable of cicatrizing.

Sometimes towards the end, *cancers of the mouth*, *larynx*, or *throat* break through to the surface, but this condition can hardly be termed a sinus. The diagnosis is easily made by the appearance of the opening on the skin and by the symptoms connected with the original diseases.

CHAPTER XXIV.

TUMOURS AND ALLIED SWELLINGS OF THE NECK.

As the neck contains a variety of organs, it offers examples of tumours of every kind. In discussing these, we purposely include certain congenital cystic structures as tumours, and at the same time embrace chronic inflammation of the lymphatic glands in this section.

Before we ascertain the nature and point of origin of a new growth, we must be quite sure that we are really dealing with a *tumour*, even in the somewhat extended application of the term as just indicated. There are morbid conditions in the neck, producing **pseudo-tumours**, which may mislead even the experienced. *Tubercular abscesses* should be especially mentioned in this connection. These start from the vertebral column, gradually track towards the surface, and have, as previously stated, often been mistaken for tumours (p. 143).

If we find a hard, slightly movable tumour in the submaxillary region, or even posterior thereto, we must think of the *board-like phlegmon*, of varied origin. The absence of fever and pain on pressure, its sharp limitation and sometimes a certain mobility of the skin over it, appear to point to a new growth. Even if the skin has become extensively adherent, the limitation of the structure is so sharp that it is difficult to discard the idea of a malignant growth. The careful consideration of all possible sources of infection will, however, lead to the recognition of the true nature of the disease. But sometimes an *exploratory* puncture only will give the first clue.

This occurred to me in the case of an aged female who had a phlegmon at the side of the neck which looked like the terminal stage of an inoperable carcinoma. Its explanation was found in a periostitis of the root of a molar, which had run its course almost unnoticed.

On another occasion I had a case which seemed to be a sarcoma of the side of the neck, and which had become adherent to the skin. It had been preceded by symptoms arising from the cranial nerves. An exploratory puncture showed that the case was probably one of *actinomycosis* starting in the sphenoidal sinus, and this was subsequently confirmed by autopsy.

Tubercular or *gummatous* deposits in *muscles*, especially in the sterno-mastoid, may be mistaken for tumour. The same applies to *œsophageal diverticula*.

We shall refer to the diagnostic difficulties presented by inflammation of the *salivary glands* and the *thyroid gland* in their appropriate place. We shall also subsequently discuss the not infrequent errors of diagnosis to which *aneurisms* and *cervical ribs* give rise.

In order to facilitate the survey we will discuss the conditions topographically.

A.—THE ANTERIOR TRIANGLE.

This triangle, particularly the space between the external border of the sterno-mastoid and a horizontal line drawn through the upper border of the thyroid cartilage, is so dominated by the *thyroid gland* that our first question in regard to any tumour in this region must be to ask whether it originates in this gland or not. *One* classical sign will seldom fail us, *i.e.*, the ascent of the tumour with the larynx and trachea on swallowing. This sign only becomes indefinite or entirely absent if the diseased gland is so firmly adherent to the adjacent structures that it actually prevents the ascent of the trachea.

Further evidence is afforded by palpation, because this demonstrates with more or less certainty the connection of the tumour with the thyroid gland. The relation of these tumours to the sterno-mastoids is

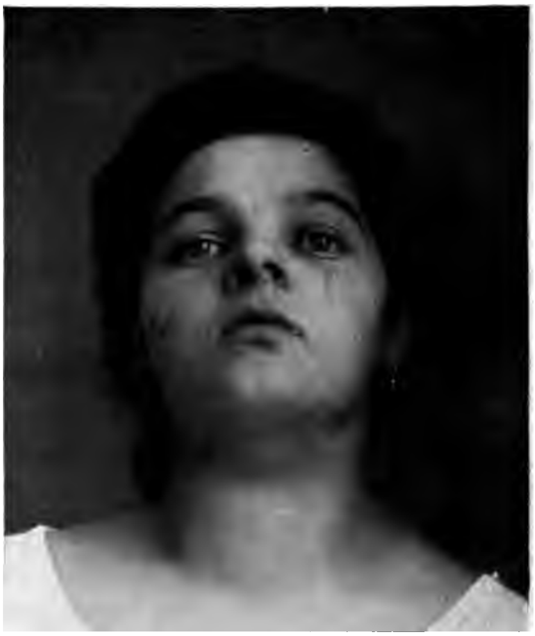


FIG. 87.—Simple hyperplasia of the thyroid gland ("full neck") in a chlorotic girl.

not at all constant. They may project forwards between them, or glide under them towards the sides of the anterior triangle, or the muscles may be spread out flat over them.

The most common disease of the thyroïd gland, a tumour in the wide significance of the term, is **goitre.**

The connection between *goitre* and the *thyroid gland* appears to us to-day to be obvious. But up to the middle of the nineteenth century the terms *bronchocele* and *air-goitre* (Luftkropf) prevailed in German literature, thus showing the predominance of imagination over observation. At the end of the eighteenth century the theory was maintained that a goitre was a hernia of the air-passage, and the term bronchocele still persists in English as a vestige of this error. In the Middle Ages and up to within the last hundred years there was a general confusion between goitre and lymphatic gland enlargement. A vestige of this error still remains in the use of the term "struma" for "tubercle," which was formerly common in France and still is in vogue in England.

I have called a goitre a tumour in the "widest sense of the term," because most goitres are not tumours in the pathological sense, but are processes of hypertrophy, hyperplasia and degeneration, often associated in striking confusion with changes which possess certain characteristics of tumour formation. The external appearances of the goitre afford some indications of all this. However, to ascertain *which* variety of goitre we are dealing with is not a mere whim, but is a matter of therapeutic significance.

(1) THE EXTERNAL APPEARANCES OF GOITRE.

We have in the first place to distinguish between (*a*) the *diffuse*, and (*b*) the *circumscribed* or *nodular* goitre.

(a) Diffuse Goitre.

The **diffuse goitre** (fig. 88) imitates in considerable measure the horse-shoe shape of the normal thyroid gland, and is of fairly uniform consistence throughout. If it feels soft, like a normal thyroid gland, and there are no accompanying vascular symptoms, the case is one of simple *hyperplasia*, without any other histological changes, except, perhaps, a slight enlargement of the vesicles. Such a goitre hardly grows beyond three times the size of the normal thyroid gland. It usually occurs in young, and especially chlorotic, girls in the period of adolescence. If the goitre is rather firm, and feels somewhat nodular, the case is one of *diffuse colloid goitre*, in which the vesicles are greatly dilated, the colloid material increased, and under pressure. If the tumour is softly elastic, compressible, and has an expansile pulsation, and if on auscultation all kinds of blowing murmurs, appreciated by the hand as thrills, are heard over the large thyroid vessels as well as over the goitre, the case is one of *vascular goitre*.

It may be noted here that the superior thyroid artery can easily be felt on the external surface of the thyroid cartilage, whereas the inferior thyroid artery recedes from the finger, owing to its deep situation. In examining for pulsation, one must be careful not to mistake the beating of thé carotid artery for expansile pulsation, and, of course, care must be taken not to look upon a burrowing abscess beneath a forwardly displaced carotid artery as a pulsating goitre—as has actually occurred.

If we are confronted by a vascular goitre, we should instinctively look for the other symptoms which go to make up **"Graves's disease."**

If the whole symptom-complex is well pronounced—a pulsating goitre, exophthalmos, tremor, and tachycardia—even the most inexperienced can have no doubt about the diagnosis. The exophthalmos, more than anything else, would indicate the diagnosis to him.

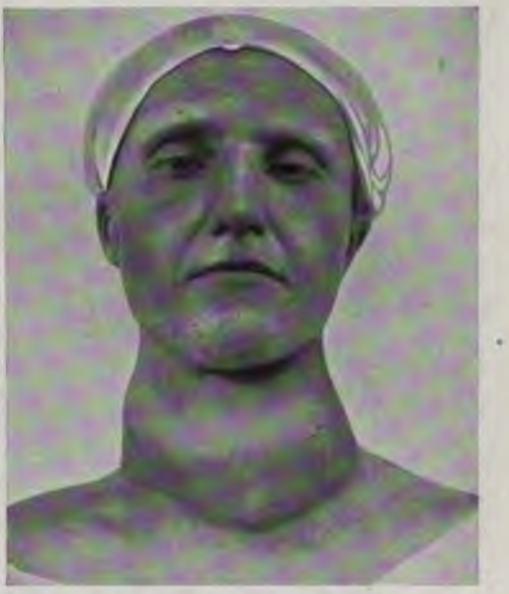


FIG. 88.—Diffuse colloid goitre.

If there be leisure for further diagnostic exercises, a search may be made for the rarer symptoms. As far as the eyes are concerned, some of these are due to mechanical causes, and others to muscular weakness — for instance, the width of the palpebral fissure, the fixity of the lids (Stellwag), deficient movement of the upper lid on looking upwards and downwards (Gräfe), insufficiency of convergence (Möbius). Genuine ocular paralyses, which are occasionally met with, must not be confused with these symptoms.

Is there anything in the form of the goitre in favour of, or against, Graves's disease? This evidence is only circumstantial. In Graves's disease the goitre is diffuse and vascular (figs. 89 and 90). Histologically a diminution of colloid substance can be demonstrated, and a proliferation of epithelium. But any variety of goitre may become "Graves's disease" secondarily. We must therefere not conclude that a case is not Graves's disease because the patient has a nodular goitre.

Easy as the diagnosis is, when the classical triad of symptoms is present, it may be extremely difficult when the disease runs an irregular course. The clinical picture presented by the disease is, however, more frequently atypical than typical, and a large number of subordinate varieties have been described—to the great confusion of students, and even of experts. If we rigidly cling to the view that hyper-thyroidism is the cause of most of the symptoms of Graves's disease, it is not always easy to distinguish between the normal and pathological, because transitory anomalies of function are conceivable in the thyroid, as in other glands. We should therefore limit the designation of Graves's disease to symptoms of hyper-thyroidism which remain permanent.

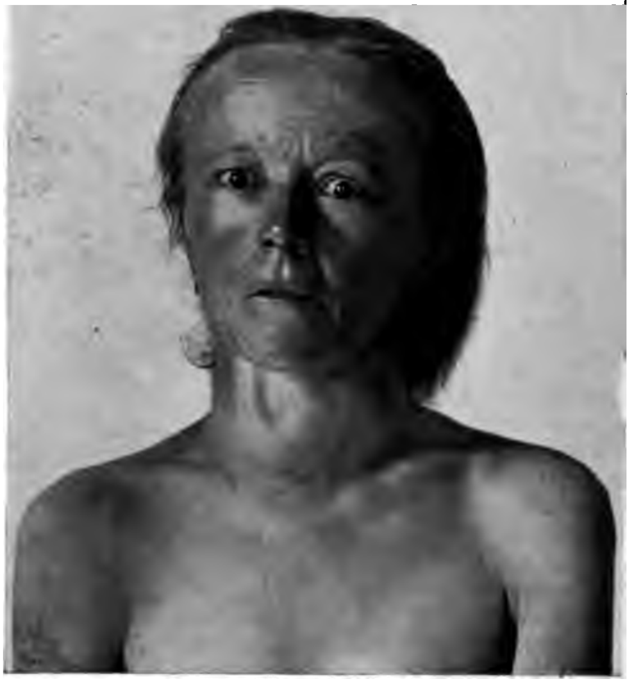


FIG. 89.—Early stage of Graves's disease.

The clinical picture remains incomplete in many cases for months, and even for years. The patient complains of emaciation, increasing muscular debility, tremors, slight perspirations, unexplained diarrhœa, long before any exophthalmos indicates the diagnosis to the practitioner. If such a patient, when completely at repose, presents strong pulsation of the carotids, it is as definite an evidence in favour of

Graves's disease as a normal amplitude of the carotid pulse would be against that diagnosis.

The instability of the psychical equilibrium of the patient is frequently so predominant that the case may be treated for years as one of neurasthenia or hysteria, until a careful examination reveals the cause of the nervous disturbances. In other cases, the cardiac symptoms are the most predominant, and the goitre itself attracts little attention, as long as no other symptoms of Graves's disease have appeared. Whether these cases should be regarded as thyro-toxic goitrous hearts, or as examples of the "forme fruste" of Graves's disease, is merely a matter of terminology.

It is obvious that we must not confuse circulatory disturbances, which are caused by the mechanical effect of the goitre, with cardiac disturbances, which are of toxic origin. I admit that the distinction is not always easy. Finally, one must not conclude that every case of prominent eyes is one of Graves's disease. Prominence of the eyeballs may be a family peculiarity. In order to establish the diagnosis of Graves's disease, it is necessary to show that the previous position of the eyes was normal, for which purpose we can derive assistance from earlier photographs of the patient.

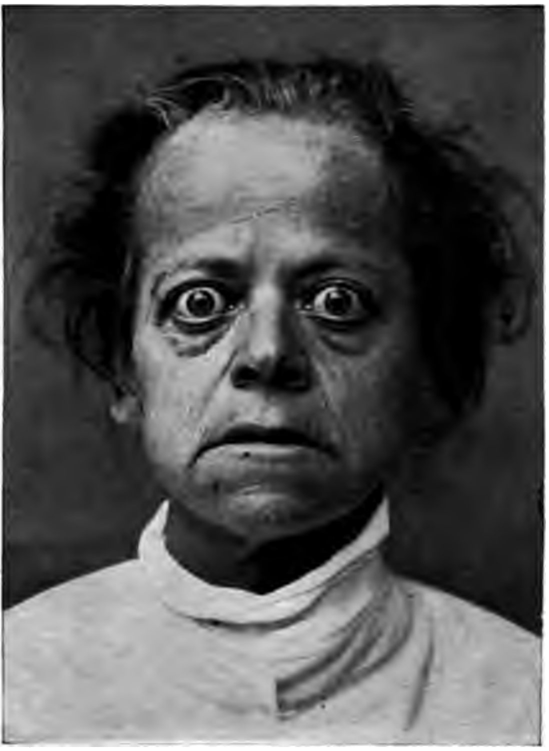


FIG. 90.—Pronounced Graves's disease.

The examination of the blood is of interest for the diagnosis of Graves's disease, although this is rather a matter for the laboratory than for the consulting room. According to Kocher and others, Graves's disease is characterized by a decrease in polynuclear leucocytes, an increase of lymphocytes, large mononuclears, and eosinophiles, and by a decrease in coagulability.

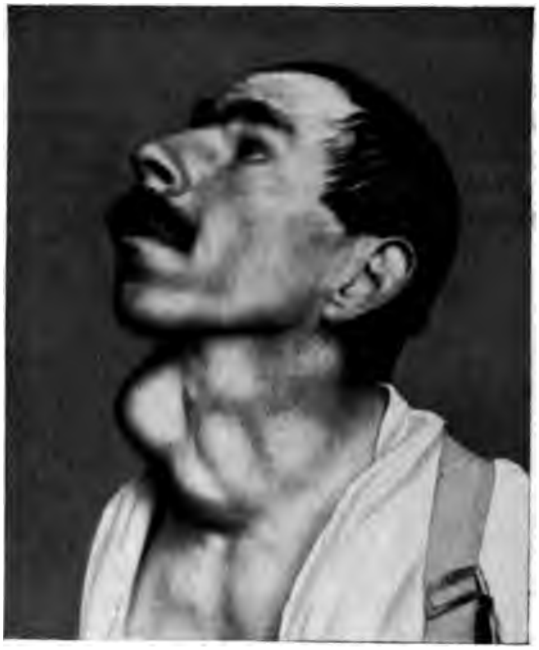


FIG. 91.—Colloid goitre, mainly of right side, consisting of separate lobules.

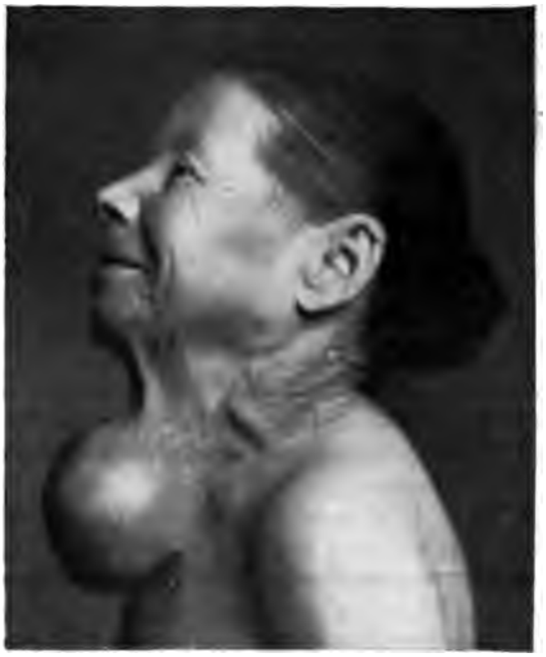


FIG. 92.—Pendulous goitre.

Similar changes occur in ordinary goitre, and even in hyperthyroidism; but in the latter the coagulability is often increased. The interpretation of the blood-picture therefore requires great care. Our own experience seems to show that more importance attaches to a pronounced increase in the antitryptic content of the blood, but the demonstration of this cannot be expected from the general practitioner. Nevertheless, the employment of this test is desirable in doubtful cases, because of the prevailing fashion of diagnosing "Graves's disease" on insufficient evidence. Not every goitrous individual who trembles occasionally is the subject of Graves's disease.

Recent researches have shown that certain relations exist between Graves's disease and changes in the thymus; but no useful assistance from the point of view of diagnosis has hitherto followed therefrom. Still less has this followed from the more or less hypothetical relations which have been stated to exist between the thyroid and other glands, with internal and external secretions.

If an elderly person is suffering from symptoms of Graves's disease, we should always inquire whether iodine has been taken. The rapid disappearance of goitrous tissue after iodine treatment leads to a flooding of the system with thyroid gland products, which manifest themselves, especially in old people, by symptoms of Graves's disease, persisting for months.

(b) Circumscribed Goitre.

In the circumscribed goitrous degeneration of the thyroid gland, **nodular goitre,** the entire parenchyma is more or less damaged—at any rate histologically—but the appreciable changes are limited to a few places which grow into nodules. These nodules are at first composed partially of tissue rich in colloid material, and partially of hyperplastic and adenomatous-like proliferation ; they may secondarily liquefy and become cysts.

The cysts which arise from dilatation of individual vesicles (giant vesicles) have no clinical significance.

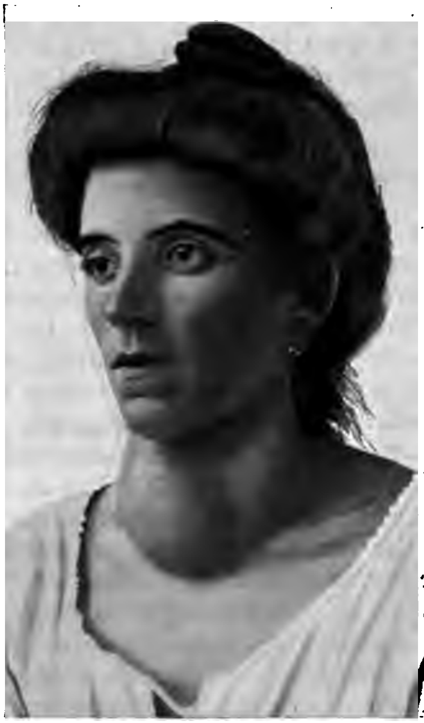


Fig. 93.—Cystic goitre.

Frequently there is only one nodule—when it is of an extensive size. In other cases, especially in elderly people, there is a whole bunch of goitrous nodules in the thyroid gland, one exceeding another in circumference. If a nodule is regular in form, smooth on its surface, softly or tensely elastic in consistence, it may be a *hyperplastic* (*follicular*) *nodule*, *a colloid nodule* or a *cyst*. Purely hyperplastic nodules are usually small, and their clinical significance is slight. Pure colloid nodules rarely reach the size of a fist. The differentiation of a cyst of smaller size than this is often impossible clinically, because they do not show fluctuation, but are softly elastic, just like colloid nodules.

A sudden increase in size and the onset of tension points to hæmorrhage within the cavity, and justifies the diagnosis of a cyst.

The distinction, however, between nodules and cysts is not of great therapeutic importance, because nobody treats cysts now by puncturing them or by opening them in the old-fashioned way, like an abscess, and allowing them to shrivel up gradually.

Firm or hard portions indicate fibrous degeneration or calcification, as can easily be demonstrated by Röntgen rays. This change may occur both in colloid nodules and in cysts, especially in old people.

There is an intermediate form between the diffuse and nodular goitre, in which the whole thyroid gland has become converted into a conglomeration of small colloid nodules.

(2) POSITION OF THE GOITRE.

The *point of origin* and the *position of the goitrous nodule* have their significance. It usually arises from one of the lateral lobes and but seldom from the isthmus, even when it appears to be just in the middle of the throat. The sterno-mastoids cause it to follow this route. If it goes on growing in this position, it will finally become a *pendulous goitre*, a variety which is exceedingly rare in these days of operations for goitre. Less frequently the nodule glides right under the sterno-mastoid and appears in the inferior lateral triangle of the neck. But more dangerous than the goitres which make their way outwards are those which remain hidden under the sterno-mastoid or make their way towards the aperture of the thorax instead of externally. If a goitre is drawn into the thorax on deep breathing, or is displaced therein by contraction of the sterno-mastoids, it is spoken of as a *wandering goitre*, a *sunken goitre*, *goitre plongeant* (figs. 94 and 95). It is quite easy to make such a goitre evident by swallowing, or making the patient droop his head so as to relax the sterno-mastoids. A goitre which is visible and palpable in the neck, but whose lower extremity is so far within the thorax that it cannot be reached except by means of the manœuvre just mentioned, is called a *deep goitre*. A goitre situated mainly or entirely in the thorax is called an *intra-thoracic retro-sternal goitre*. We shall discuss this in connection with mediastinal tumours. The extent of a deep or intra-thoracic goitre within the thorax can be fairly well estimated by *percussion*, and quite accurately determined by a *skiagram* (fig. 139). Diffuse goitres sometimes penetrate behind the trachea (*retro-tracheal goitre*) or behind the œsophagus and pharynx (*retro-visceral goitre*) (fig. 98) and cause the posterior pharyngeal wall to bulge like a retro-pharyngeal abscess. We should especially think of these varieties if the difficulties of

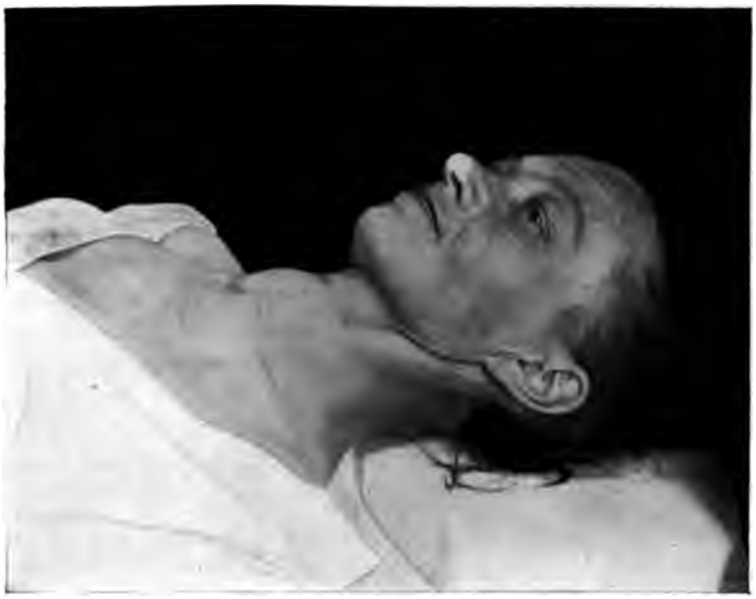


FIG. 94.—Sunken goitre with relaxed sterno-mastoids.

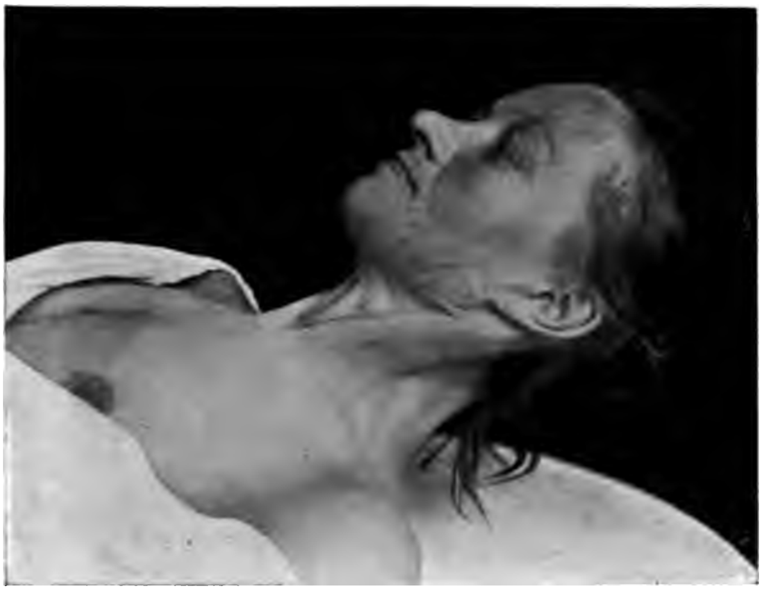


FIG. 95.—Sunken goitre pressed deep down by contraction of sterno-mastoid.

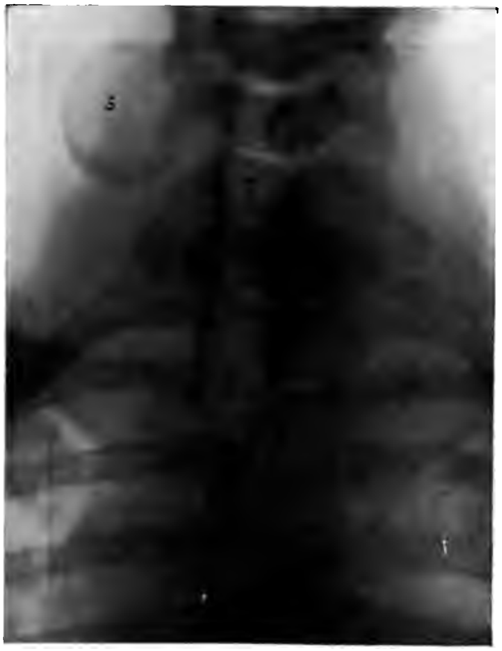


FIG. 96. –Skiagram of goitre (posterior view). S : calcified cyst. T-T, skiagram of trachea. Slight concavity on right.

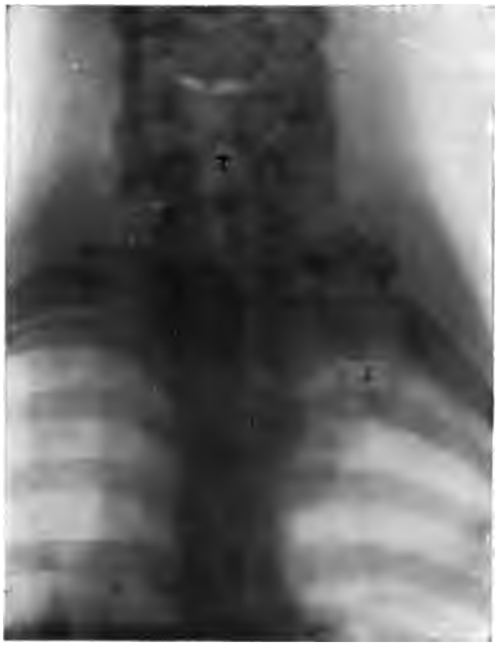


FIG. 97.—Skiagram of goitre. Severe compression of trachea on left. S calcified colloid nodule on right.

breathing and swallowing are not clearly explicable by the palpable part of the goitre.

If both sides are affected, it is important for the purpose of operation to know definitely from which side the trouble mainly arises. As a rule it is not the side which is blamed by the patient, and frequently by the beginner also; in other words, not the side which is the more projecting. Alternate pressure on both sides of the goitre, and especially the raising of the one lobule and the other from the trachea where this is feasible, will very often show whence the difficulty comes. A laryngoscopic examination, of course, yields more definite information. It not only shows the lateral displacement, the twisting and bending of the larynx, but also the convexity of one tracheal wall or the flattening of both walls. A skiagram demonstrates the entire condition of the trachea even more clearly, and one should be taken in every case which is not quite clear. It may then be possible to tell the patient before the operation that it is not the externally visible goitre which is to be removed, but a much more dangerous one, which is concealed.

In discussing the anomalous positions which goitres may occupy we have hitherto assumed that the abnormally placed goitre is directly connected with the thyroid. As a matter of fact, this is generally the case; but the goitre may have developed in an accessory thyroid gland. These are termed *secondary goitres*, as suggested by Wolfler. They are called *genuine* if they have no anatomical connection with the main gland, and *false* if they are connected with it by means of a bridge of tissue. Lingual and tracheal goitres are always secondary goitres, sometimes also the intra-thoracic variety, but the other forms, such as retro-visceral, are rarely so.

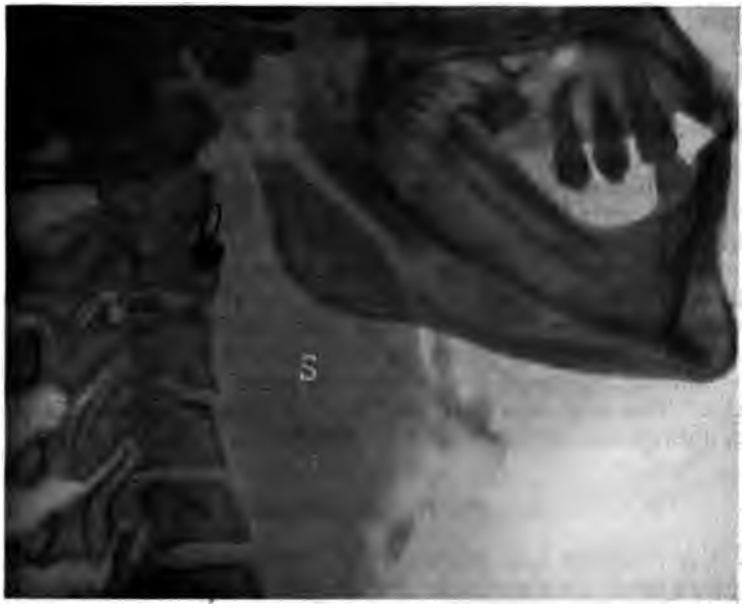


FIG. 98.—Skiagram of retro-visceral goitre (S).

(3) COMPLICATIONS OF GOITRE.

HÆMORRHAGE, INFLAMMATION AND MALIGNANT DEGENERATION.

The *complications* of goitre possess diagnostic interest as well as the condition itself. These complications consist of *hæmorrhage, inflammation* and *malignant degeneration.*

(a) Hæmorrhage.

A patient suffering from a colloid or cystic goitre may suddenly, or sometimes overnight, be seized with rapidly increasing dyspnœa, combined with a feeling of tension in the goitre and visible enlargement thereof, with moderately severe pains radiating towards the jaw, back of the neck, ear and shoulder. These symptoms may come without any external cause, or after an injury, or congestion of the circulatory system through coughing or vomiting. They reach their maximum in a short time, then they remain at a standstill, and subsequently decrease gradually. This assemblage of symptoms points to **hæmorrhage.** The goitrous nodule is found to be tensely stretched, even hard, somewhat painful on pressure and, if not of great circumference, quite movable. This mobility enables us to exclude forthwith any inflammatory process or malignant goitre with similar symptoms.

A colleague of mine watched his own goitre in a mirror enlarging until severe dyspnœa set in. He made the diagnosis and drove off at once to the surgeon. Meanwhile the hæmorrhage had attained its maximum and no interference was required,

I once operated on a young girl whose goitre increased during a pleasant evening's walk from unnoticeable dimensions to that of a medium-sized apple. A little morphia calmed the dyspnœa, and the operation which was done immediately showed that the nodule was tensely filled with blood.

On another occasion I was consulted about a young girl with malignant goitre. The diagnosis had been based by an experienced practitioner on its hard consistence, radiating pains and recent increase in growth. The nodule was, however, too movable to attribute the radiating pains to malignant goitre, and further the pains and enlargement had come in attacks within the last two months. The case was therefore one of cystic goitre with hæmorrhage, a diagnosis confirmed by the operation.

(b) Inflammation,

If the swelling and difficulty in breathing and swallowing do not reach their maximum in the course of minutes or hours, but only after a day or two, and if there exist also severe pain on pressure, sharp local and radiating pains, adhesion to the skin and the deeper organs, and more or less high fever from the beginning, the case is not one of hæmorrhage into the goitre, but of inflammation thereof—an **inflamed goitre.** If the skin is œdematous and red, and fluctuation is present, or if pus bursts through eventually—rarely into the trachea or pharynx—the state of affairs is obvious.

Timely intervention by operation nowadays renders it impossible for a suppurating goitre to burst into the trachea; but even without operation this is a rare contingency. But on the other hand the pressure of the goitre on the trachea, or the inflammatory œdema of the laryngeal mucous membrane, may lead to dangerous dyspnœa or even to suffocation.

Peter Frank, who wrote on this subject a century ago, describes the case of a 7-year-old lad who was nearly suffocated by a suppurating goitre. The village quack said that a nerve in the vicinity of the larynx was torn and that a fatal result was unavoidable. The mother made a more correct diagnosis, and she implored the local barber to open the abscess between the swollen veins. The desired result followed.

A practitioner in the middle of the last century describes how he allowed a young man to die from gradual suffocation of an inflamed goitre, bemoaning his sad fate, but without the energy to venture on the life-saving incision.

An inflamed goitre is always of a metastatic nature, and comes on especially after scarlet fever, typhoid, or puerperal fever, but also after trifling derangements of the trachea and œsophagus—sore throat or intestinal catarrh, &c. This fact may be important in differentiating between this condition and hæmorrhage. The bacteriological examination of the pus has made it possible to establish the diagnosis of a recent attack of typhoid.

If the swelling has arisen in the course of a catarrhal affection of the respiratory tract, there may be some doubt about the diagnosis. The infection would point to an inflamed goitre; the increased pressure through coughing would suggest hæmorrhage. No importance is to be attached to a slight rise in temperature, because this may occur in hæmorrhage, quite apart from the catarrh. The differentiation depends upon the whole course of the symptoms and upon the local findings. If the tumour is immovable and there is considerable local heat and pain on pressure, the case is one of inflamed goitre, notwithstanding that the other symptoms may be in accord with the diagnosis of hæmorrhage.

The *normal* thyroid gland may inflame just as a goitre. This usually occurs after infectious diseases, especially typhoid, malaria, influenza and articular rheumatism, but may occur as a *clinically primary* disease. This is not an inflamed goitre, but a *thyroiditis*—a condition insufficiently recognized. Just as in the case of an inflamed goitre, this does not always lead to suppuration, but may resolve in the course of a few days. This is the form which follows acute rheumatism, malaria and influenza, and is also the clinically primary form. It is best termed, as Mygind suggested, *simple thyroiditis*. It is not always easy to decide whether there is any suppuration, and if spontaneous resolution occur it does not by any means signify that there was no pus.

In a case reported by Breuer as one which ran its course apparently without suppuration, I found at the autopsy, seven months afterwards, a small abscess with inspissated staphylococcal pus.

Suppuration can as a rule be diagnosed by attention to the temperature chart and to the increasing adhesion of the gland to the adjacent organs, which circumstances show the necessity for early surgical intervention.

Sometimes the inflammation travels over the whole gland in the course of a few weeks, and finally attacks the pyramidal lobe, when the inflammation of the lateral lobes has gone down. Recurrences also occur, especially in simple thyroiditis.

There is one important differential sign, in the initial stage, between an inflamed goitre and thyroiditis, apart from the history of the goitre. This depends upon the fact that the swelling in an inflamed goitre is usually limited to one individual lobule, but nevertheless may attain a considerable circumference, whereas in thyroiditis a whole lobe, if not the whole gland, is affected, and still the swelling does not exceed the size of a goose's egg. The extension of the inflammation over the whole of the thyroid gland indicates thyroiditis.

(c) Malignant Degeneration.

Inflamed goitre and **malignant goitre** are occasionally mistaken for one another, and errors of diagnosis are made in both directions. I have seen cases of indefinitely outlined swellings of the thyroid, with redness of the skin and pyrexia, and with rapid growth, declare themselves as sarcomata at the operation. On the other hand a chronic inflammation in an old, hard goitrous lobule may present all the clinical signs of malignant growth, including even metastases and a fatal result. It is quite impossible to distinguish between such rare conditions as **syphilis** and **tubercle** of the thyroid on the basis of the clinical symptoms. If the clinical course, the history, and a positive Wassermann reaction suggest syphilis, and if the symptoms are not urgent, a trial of specific treatment is indicated. Otherwise, any suspicion of malignancy demands operation without delay. Hitherto, tubercle has been first discovered at the operation, or more correctly speaking has been recognized on histological examination, there having been no clinical symptoms.

A malignant goitre can usually be confidently diagnosed from the following signs :—

(*a*) An unexplained steady growth of a goitrous nodule in a patient over 30 years of age.

(*b*) The onset of hoarseness not accounted for by the size of the tumour (paralysis of the recurrent laryngeal).

(*c*) Radiating pains towards the jaw, ear, back of neck and shoulder without any acute inflammatory symptoms or signs of hæmorrhage.

(*d*) Diminution of mobility, irregularity and unevenness in form, and hard consistence of the goitre.

As long as the malignant degeneration is limited to the interior of the goitrous lobule, the unexplained growth is its only striking feature. There is neither hoarseness nor radiating pain. Nothing short of the microscope can yield a positive diagnosis.

On the other hand, a contracting cancer and even a sarcoma, in consequence of early adhesions with adjacent structures, may cause hoarseness, radiating pains, possibly also narrowing of the pupils and other signs of sympathetic paralysis, while the growth itself has not yet attracted the attention of the patient and may really require looking for.

Operation must never be delayed until all the signs of malignancy are present, because the aim is not diagnosis but cure. The prognosis in malignant goitre is only favourable as long as the growth is within the capsule of the goitre, and therefore must be suspected rather than diagnosed.

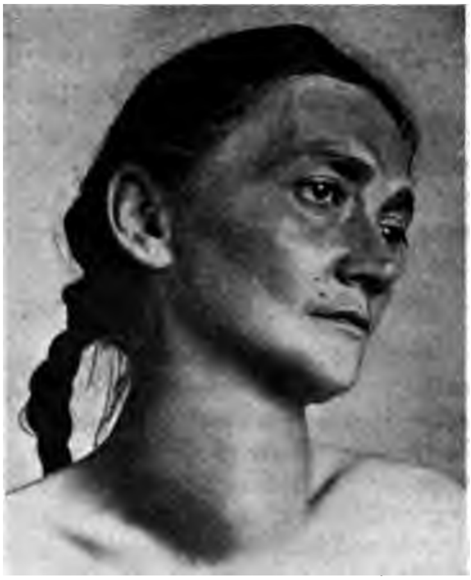


FIG. 99.—Malignant goitre.

The distinction between *sarcoma* and *carcinoma* need not detain us, because even the interpretation of the histological appearance is not always easy. The differentiation between the various forms of carcinoma appears to be of even less practical importance so far. But it should be observed that the adenomatous variety (the "proliferating goitre" of Langhans) runs a remarkably slow course, despite the presence of all the signs of malignancy.

On one occasion I declined a further operation on a female patient suffering from paralysis of the recurrent laryngeal, with extensive adhesions and thrombosis of the veins of the neck, and informed the friends that the course of the disease would be rapid. The patient, however, lived for nearly nine years in great suffering.

The *metastatic growths* from thyroid gland tumours have certain special characteristics which are also of diagnostic interest.

Colloid goitres, which are clinically and histologically apparently innocent, sometimes give rise to metastases, especially in the bones, with a structure partly composed of normal thyroid tissue, partly of colloid goitre and partly of cancer.

Sometimes the metastases of definite thyroid gland cancers consist of normal thyroid tissue by way of atavism.

All forms of cancer of the thyroid have a predilection for metastases in the osseous system.

Growths independent of the thyroid gland which occur in the anterior triangle of the neck are rapidly disposed of. They are *branchial cleft cysts*, or, when situated in the median line in front of the thyroid cartilage, they are cysts of the *thyro-glossal duct*. We shall discuss these in connection with the other cysts of the neck.

B.—TUMOURS OF THE SIDE OF THE NECK AND ITS VICINITY.

The **tumours** which are found at the **side of the neck** differ widely in character, and in order to avoid repetition we shall include among them new growths under the sterno-mastoid and those which occur in the submaxillary and parotid regions.

(1) ENLARGEMENT OF THE LYMPHATIC GLANDS.

In the first place we must be quite sure that we are really dealing with **enlargement of the lymphatic glands.** These always declare themselves by their number and their arrangement in groups. If a tumour, which is apparently single, is really made up of separate lumps or nodules, we must think of a mass of adherent glands. Appreciable swelling of one individual gland may occur, both in tubercle and in secondary malignant degeneration, but is not common in either.

If we recognize the tumour to be of glandular origin, it may be due either to inflammation, or to new growth, or to the intermediate condition of malignant lymphoma.

I am not treating now of acute inflammation of the cervical glands. Their behaviour is similar to that of the abscesses to which they give rise, to the description of which the reader is referred.

If a child suffering from eczema or a chronic mucous membrane catarrh has enlarged glands in the area drained by the corresponding lymphatics, the diagnosis is simple **lymphadenitis.** We must assume that the glands have been affected by the constant introduction of mildly virulent organisms or by their toxins.

Formerly, simple and tubercular lymphadenitis were included under the one term "*scrofula*." To-day this term has no meaning, unless we reserve it to express that general state of lowered resistance which is due to hereditary causes (alcohol and syphilis), and to unfavourable hygienic surroundings, and which prepares the soil for various micro-organisms. If the tubercle bacillus be among these—a very frequent circumstance—then scrofula becomes tuberculosis. Adolf Czerny and his school apply the term "exudative diathesis" to any condition wherein there is hyper-sensitiveness to infective organisms, and they only speak of "scrofula" if a tubercular infection is super-added to this diathesis.

Even the beginner knows that a chain of nodules which are partly elastic and movable, partly hard and infiltrated or even fluctuating, are **tubercular.** Scattered scars indicate to him that the disease is not recent, and that there is no reason to entertain the idea of malignancy (fig. 100). If there be but little pain, and the mobility is free, and the consistence is elastic with no sign of involvement of the skin despite their long existence, we know that the glands are of the non-caseating, or of the very slowly caseating variety. If there be definite pain on pressure, with only slight mobility, the consistence firm, and the superjacent skin can only be picked up with difficulty, it shows that caseation is proceeding and that peri-adenitis has begun. If the skin has become red, and the glands are fixed and their firmness is replaced by a softly elastic or fluctuating consistence, there can be no further doubt about their suppuration.

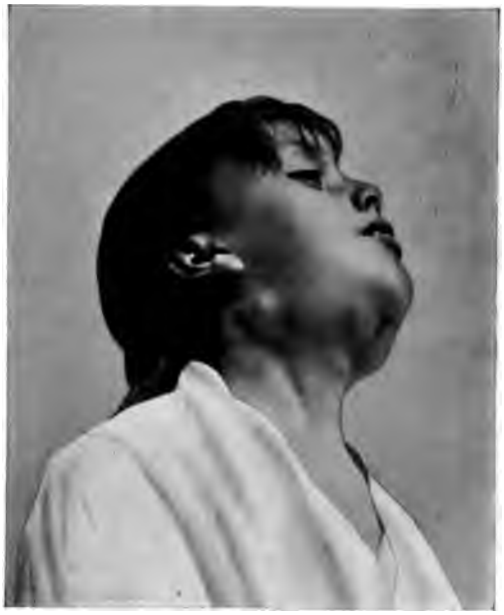


FIG. 100.—Tubercular lymphoma.

If a patient who has hitherto been free from glands, acquires a bunch of them on one side, within a short time, one must think of the possibility of a **syphilitic** infection, and a primary sore must be looked for in the area drained by the corresponding lymphatics, especially in the mouth or nose.

Sometimes it is difficult to distinguish between tubercular glands and the large glands which occur in the course of such lymphatic diseases as leukæmia and lymphadenoma. **Leukæmia** is easy to recognize if the glandular enlargement is overshadowed by such signs as pallor, debility, and the hæmorrhagic diathesis—but an examination of the blood should be made in all doubtful cases to settle the diagnosis.

Glandular enlargements due to pseudo-leukæmic conditions are more difficult to differentiate.

We adhere to the view that *pseudo-leukæmia* (malignant lymphoma, Hodgkin's disease) is a clinical unity, because the progress of the disease, with its invariably fatal termination, in spite of remissions, displays a unity of character in its main aspects, and does not permit of distinct separation into two or more groups. Histological examination does, however, suggest that this disease, which from the clinical standpoint is apparently one, may in reality represent two different conditions, anatomically, and, perhaps, also ætiologically. The one variety is *Cohnheim's* (aleukæmic lymphomatosis, lymphocytoma), recognizable by a simple increase in lymphadenoid tissue, and the other is *Lympho-granulomatosis* (malignant granuloma of Paltauf, Beuda, Sternberg, &c.), recognizable by the presence of a peculiar granulation tissue in all lymphatic structures. Necrotic changes are present in both varieties, but more frequently in the latter, while suppuration occurs in neither. In the former variety there is an increase of lymphocytes in the blood, in the latter the increase concerns the polymorphonuclear neutrophile leucocytes—but these changes are not constant. The ætiology of both varieties is obscure. They have nothing to do with tubercle, despite occasional association. The process is a much more malignant one, and certainly is not an "attenuated" tuberculosis. It is quite possible, however, that the variety known as lympho-granulomatosis may be due to an organism allied to the tubercle bacillus, but of a much more malignant character (bacillus of Much and Frankel).

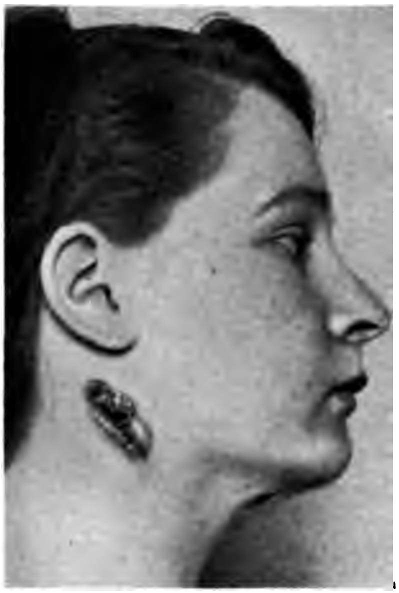


FIG. 101.—Tubercular changes of the skin in case of tubercular glands.

The simplest cases to diagnose are those wherein all the lymphatic glands of the body are enlarged. Such a universal enlargement does occasionally occur in tubercular glands, but very much more rarely than in pseudo-leukæmia.

In the initial stage when the neck alone is affected, and therefore the extension of the glandular enlargement offers no points of differentiation, the diagnosis may be indeed difficult. Our opinion must be based on the size and condition of the glands and their relation to the surrounding parts. If they are of medium size, soft and immovable, it is, unfortunately, impossible to dogmatize about the diagnosis, because a non-suppurating form of tubercular lymphoma cannot at this stage be distinguished from the soft variety of malignant lymphoma. In such cases the latter is always first recognized by its generalization, or by excising a portion for examination.

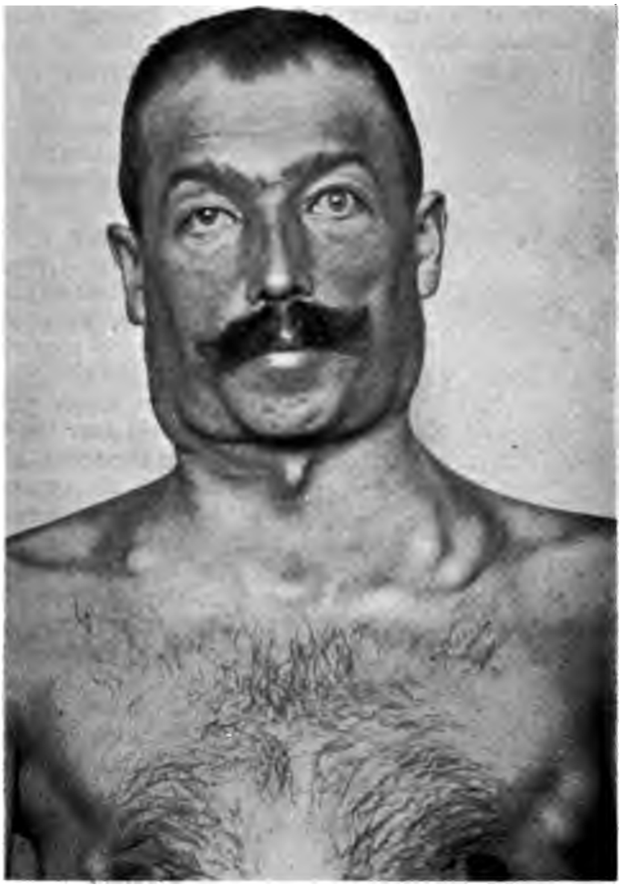


FIG. 102.—Leukæmic lymphoma of neck and axillæ.

It is quite different if numerous movable, but hard, glands are present. Such a condition indicates the hard form, or rather the hard stage, of Hodgkin's disease. Tubercular glands only exhibit the pronounced hardness, which the latter disease occasionally manifests, after a recent inflammation. But in such cases signs of peri-adenitis, and especially of adhesions with the surrounding tissues, are present.

It must not be supposed that the glands cannot become pretty fairly adherent in lymphadenoma. Indeed, adhesions do occur, but they are not accompanied by as much peri-adenitic infiltration of the tissues as occurs with caseating tubercular glands. Although the glands may be very hard in lymphadenoma, their outlines can be distinctly felt, and this serves to differentiate it from tubercle. The glands are also more prominent, so that the whole aspect resembles a caricature of tubercular adenitis.

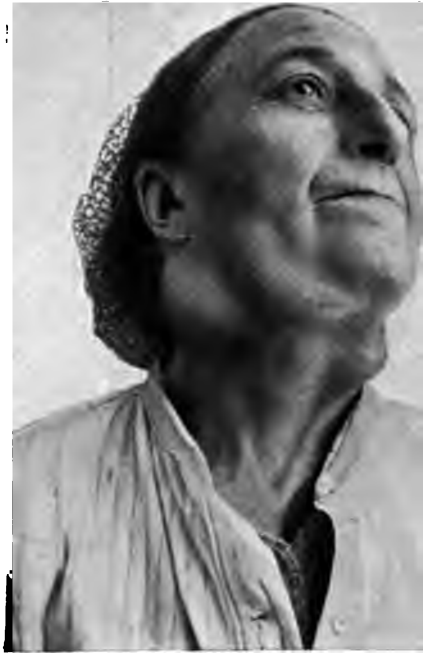


Fig. 103.—Lymphadenoma (pseudo-leukæmia). Early stage.

There is one rather rare symptom which is in favour of lymphadenoma (and leukæmia), and against tubercle, as showing that the bone marrow participates in the disease, and that is, severe pain on percussion over the sternum.

If the mediastinal glands are at all extensively enlarged, it is very suggestive of pseudo-leukæmia (and leukæmia) rather than of tubercle, although this enlargement may exist, as a rare phenomenon, in the latter. A rise of temperature is common in both conditions. In pseudo-leukæmia this rise is higher than in uncomplicated tubercular glands — and may comprise entire weeks of pyrexia.

Blood examination is not at all conclusive. The slight anæmia of malignant lymphoma, can just as well accompany tuberculosis; neither is the leucocyte content of the two conditions typically different. But as the treatment and prognosis of the two diseases differ so greatly, it is advisable to excise a portion of a gland in all doubtful cases, for examination, which will also permit of the differentiation of the two forms of pseudo-leukæmia—a differentiation which may eventually possess some therapeutic importance.

Malignant enlargement of glands is especially indicated, when they are hard, contract early adhesions to surrounding parts, and cause radiating pain. It is often very difficult to demonstrate the primary tumour, because it frequently bears no relation in size to the extent of the glandular enlargement and may, therefore, lie in a very concealed situation.

I once found a small carcinoma at the hinder end of the middle turbinate bone (fig. 105), as the primary growth in a case of considerable enlargement of the glands at the side of the neck. In another case the primary growth was at the posterior border of the septum nasi.

A growth in the neck can only be regarded as primary, if a careful search has excluded cancer from the various sites of the internal mucous membrane which are usually attacked.

If the cancerous infiltration has gone beyond the glands, and has penetrated the skin and become secondarily infected, it is very liable to be mistaken for a chronic inflammatory process (fig. 106).

On the other hand, there are certain very rare cases wherein cancerous glands have an elastic consistence and are very late in contracting adhesions. Sometimes it is quite impossible, even at the operation, to distinguish these from hyperplastic tubercular lymphomata.

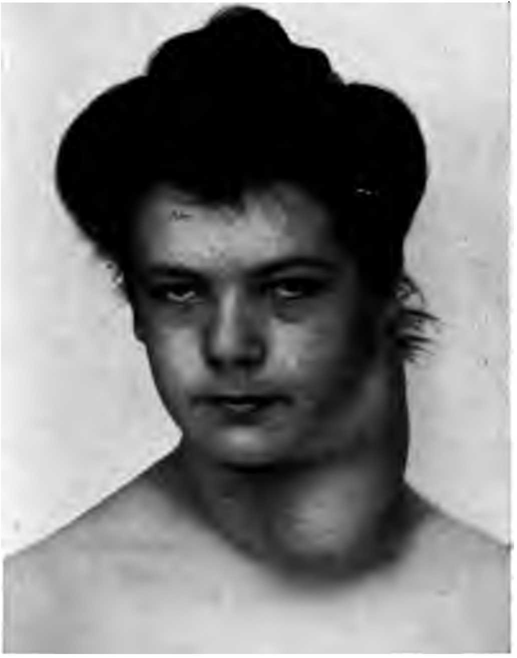


FIG. 104.—More advanced case of lymphadenoma, but still confined to the neck.

(2) TUMOURS WITH LIQUID CONTENTS.

If we have excluded disease of the lymphatic glands, and at the same time recognize that the growth is a tumour in the widest sense of the term, further deductions must be based primarily on its *consistence.*

If a tumour at the side of the neck is soft or elastic in consistence, or if it fluctuates, we must conclude that its contents are fluid. We can only be misled by a *lipoma*, which also has a soft or elastic

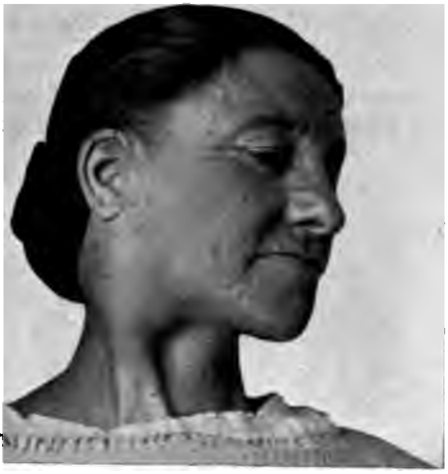


FIG. 105.—Cancerous glands, with small primary growth on the middle turbinated bone. (From the Surgical Clinic at Berne.)

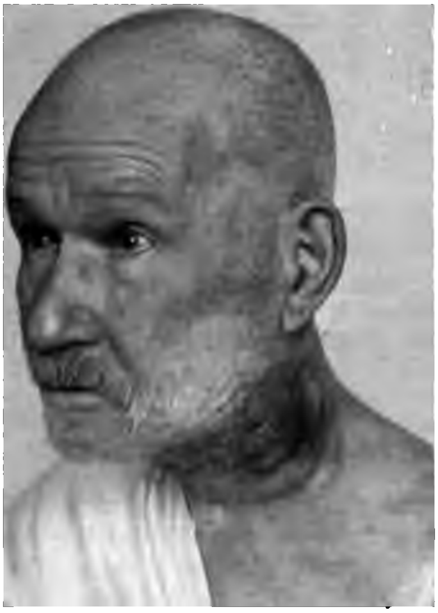


FIG. 106. Diffuse carcinoma of neck. Primary growth on tonsil. (Resembles actinomycosis, or board-like phlegmon.)

consistence, but subcutaneous lipomata may be recognized by their lobular structure and the deep subfascial lipomata are too rare to merit early consideration.

The *liquid contents of the tumour* may consist of *lymph*, *blood* or the *product of an epithelial secretion.* The liquid is either in numerous small vesicles or within one large cavity, or both conditions may be combined. This already gives rise to a large number of varieties, which are increased by the circumstance that the blood within these tumours may be either arterial or venous.

The variety is, however, not so great, if we leave a few rarities out of account and limit ourselves to a small number of typical conditions, whose diagnosis is not difficult.

(*a*) If the growth is so near the surface that its contents gleam through, but not with the dark blue colour indicative of blood, the case is one of **lymph tumour** (*i.e.*, lymph cyst) if the growth is smooth and round, but is a *cavernous lymphangioma* if it appears to be multilocular or spongy in consistence. These tumours look reddish in transmitted light and light blue in direct light, if the skin covering is thinned out sufficiently.

In addition, lymph-containing tumours react much less to changes in states of pressure (posture of body, squeezing) than do blood-cysts, and their contents are much less displaceable.

If a growth of the neck resembles a blood tumour in colour, but has all the other characters of a lymph cyst, we must first ascertain whether it has been punctured. A puncture may sometimes cause the contents of a lymph cyst to remain permanently bloody.

We have just drawn a distinction between one-chambered lymph cysts and cavernous lymphangiomata, but there is really no sharp differentiation between the two. The most pronounced lymph cysts permeate deeply among muscles by means of cavernous strands, and many cavernous lymphangiomata contain large cystic cavities between spongy portions.

The diagnosis of cystic lymph tumours is facilitated by the circumstance that they occur in two well-defined forms, viz., *cystic lymphangioma of infants* (congenital cystic hygroma of the neck), and the *lateral lymphatic cyst of adults.* The former is present at birth, and is situated in the upper part of the anterior triangle of the neck, below the parotid region. It spreads thence, rapidly increasing in size, to all sides, and if bilateral it encircles the neck like a collar. As it approaches nearer and nearer to the skin, the latter becomes thinned out in places to a fine membrane, and at the same time the cyst penetrates deeply between the muscles and interferes with the structures in the neck, through its increasing extent. Sometimes it is possible to feel hard fibrous portions situated within the soft elastic cysts, and the whole tumour shakes like jelly on being moved.

The lymphangiomata of adults are mostly situated in the supraclavicular fossa, and occur generally in women. They consist either of large single cysts or of a conglomeration of smaller cysts.

The growth illustrated in fig. 107 consisted of one large cavity, and of a pedicle made up of dilated lymph spaces, which ran down deeply among the cervical muscles.

The purely cystic forms are liable to be confused with branchial cleft cysts, which will be discussed subsequently. In adults the lymph does not gleam through the skin as clearly as it does in children. Both growths are essentially congenital in origin, but they do not develop till later years. If the cyst is situated very much to the side, this is the only sign really valuable for the diagnosis of lymphatic cyst; positive accuracy can only be obtained by puncturing the cyst and microscopic examination of a portion of its wall.

(*b*) If the contents of the tumour look dark blue through the thin skin, the case is one of a **blood tumour.** If the surface is irregular and rough, it is called a *cavernous angioma*, and if it is round and regular it is known as a *blood-cyst*. In both varieties the contents of the cysts are definitely expressible, thus contrasting with lymphangiomata; but as soon as the pressure is relaxed the cyst immediately fills up. A deeply situated venous blood-tumour is not recognizable by its colour, but it becomes inflated when the intra-thoracic pressure is raised by coughing, crying or squeezing, or by keeping the head low. This sign distinguishes it from a deep lipoma or lymphangioma.

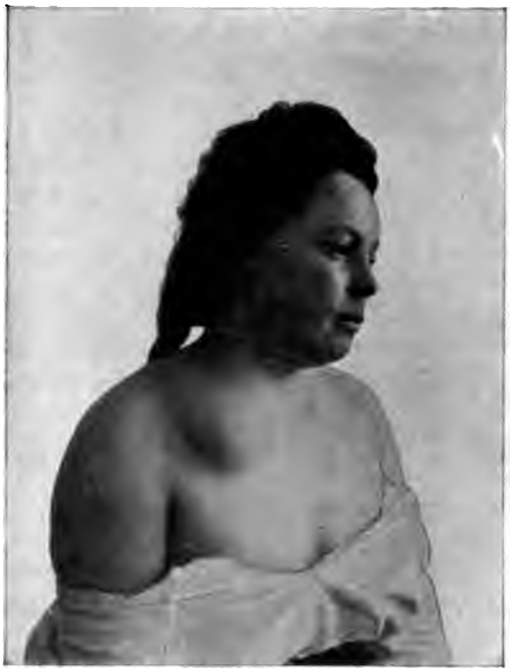


FIG. 107.—Lymphatic cyst at the side of the neck.

As we have mentioned the matter of expressibility, we must again refer to *diverticulum of the œsophagus*. Its contents can also be expressed, but it does not fill up forthwith when the pressure is relaxed. The diagnosis is readily made from the history and the fact that there is difficulty in swallowing when the diverticulum fills up, owing to pressure on the gullet.

(*c*) The *tumours containing venous blood* just referred to, proclaim the nature of their contents by their colour and expressibility; but structures filled with *arterial* blood, **aneurisms**, possess another characteristic symptom, viz., pulsation.

This feeling of pulsation may be appreciated under three different circumstances:—

(1) The tumour itself may pulsate—expansile pulsation.

(2) The tumour may be rhythmically raised by the pulsation of the carotid artery—a communicated pulse.

(3) The carotid artery itself may be pushed forward by a tumour lying beneath it.

In order to differentiate between these conditions, one must endeavour to grasp the tumour between two fingers. If these are forced apart from each other, in all situations and in all directions, the pulse is then an expansile one. If the fingers are lifted with each pulsation, but are not forced apart, the tumour is merely raised by the carotid—it belongs to the second variety. If only a pulsating strand is felt *over* the tumour, while the rest shows no pulsation, the tumour lies *under* the carotid.

If an expansile pulse really exists, the beginner is apt to think at once of an aneurism; but *very vascular sarcomata* may also yield an expansile pulse and be accompanied by loud hæmic murmurs.

Although a vascular goitre may have a pronounced expansile pulse, it could only be mistaken for an aneurism after a very indifferent examination. A vascular goitre usually affects the whole thyroid, and is, therefore, bilateral. The fact that it follows the movements of deglutition ought to remove any doubt.

The carotid artery sometimes undergoes dilatation in old syphilitic subjects at the point of bifurcation, and this may lead the beginner to think that an aneurism is developing.

If he overlooks the pulsation and neglects to examine the other side, he may regard the swelling as an enlarged gland, especially if the presence of a cancer of the lip causes him to look for one.

A communicated pulse may be observed in enlarged glands lying on the carotid artery, goitrous nodules and other tumours, especially deep-lying branchial cleft cysts.

The mistakes which occur, owing to the carotid pulsating *in front* of a pathological structure, mainly concern *burrowing abscesses* and deep *sarcomata of the neck*. Similarly a subclavian artery, pushed forward by a *cervical rib*, has often been diagnosed as an aneurism.

The *etiology* is always an important factor in the diagnosis of aneurism. Arterial dilatation is always preceded by some morbid condition of the vessel wall, produced by arteriosclerosis or syphilis, or is the result of some trauma.

If, after considering all these points, we arrive at the diagnosis of **aneurism,** we must determine the artery whence it arises. If the tumour is behind the sterno-mastoid we should think of the carotid, and if it seems to start low down its origin will probably be at the root of this artery. The delay of the pulse in the temporal artery is an important corroborative sign, but its absence is not an unconditional proof against aneurism. If the tumour is situated high up and it displaces the tonsillar region inwards, it can be nothing but an aneurism of the external or internal carotid. If the temporal pulse is weakened and delayed, we must conclude that it is an aneurism of the *external carotid*—the more frequent variety.

If an aneurismal swelling is found in the supra-clavicular fossa, there is no difficulty in diagnosing an aneurism of the *subclavian artery*. Weakening and delay of the pulse in the corresponding radial, pressure signs referable to the brachial plexus, are the classical symptoms.

It is necessary to mention a condition which is very rare in civil practice, but which is of frequent occurrence in modern warfare. In this condition the region of the common carotid presents a scar resulting from a gunshot wound, stab or cut, beneath which there is a pulsating tumour, varying in size from a waluut to a hen's egg, with hæmic murmurs audible over it. The patient complains of symptoms pointing to disturbance of the cerebral circulation, and the superficial veins of the corresponding side of the head and neck are dilated. This condition is one of **arterio-venous aneurism**, and is caused by a simultaneous injury to the common carotid artery and internal jugular vein. Exceptionally a similar condition may occur in connection with other arteries of the neck, viz., the external carotid and subclavian.

There is a difference between an *arterio-venous aneurism proper*, wherein the blood exists in a space between the artery and vein, and an *aneurismal varix*, wherein the blood flows directly from the artery into the vein. The former is somewhat irregular in outline, the latter is more circular in shape.

(*d*) A cervical cyst, which does not fit in with any of the groups just described, one which is neither a blood nor a lymph cyst—is probably an **epithelial cyst** arising from some *congenital rudiment.*

If it is situated in the middle line above the thyroid gland it arises from the thyro-glossal duct, which runs from the foramen cæcum at the base of the tongue towards the thyroid gland. A cyst in the course of this duct must be ascribed to its imperfect obliteration, whether it contain mucus or epithelial *débris.*

It might be mistaken for a swelling of the sub-hyoid bursa, a median goitrous cyst, a simple sebaceous cyst of the region, or even for an abscess in connection with the hyoid bone. But these are all rare eventualities, which can only be definitely determined by the microscope. But the history will allow us to distinguish between a cyst on the one hand, and some suppurative process on the other. Exceptionally, such a median cyst may be displaced as far as the root of the neck.

If the cyst is situated laterally, at the inner border of the sterno-mastoid, or even—in its lower portion—at its outer border, anatomical research shows that it is derived from the second, or even from the third and fourth, branchial cleft. It will contain mucus, muco-serous fluid, or fat and epithelial *débris*, like a dermoid, varying with its lining epithelium. The skin covering it is usually normal; but where it is thinned there is an absence of the translucent appearance seen in a lymphatic cyst. These cysts often stand forth quite distinctly and well defined above the level of the surrounding skin. There is one group of cases in which we can feel an oval structure lying deeply beneath a diffuse indefinite prominence of one side of the neck. If the head is turned towards the opposite side, the sternomastoid can be felt to contract over it. These tumours have been described as *deep dermoids of the neck* (fig. 108); but there is no justification for separating them from cysts of the branchial cleft. Their origin from the pharyngeal epithelium is betrayed by the abundant lymphoid tissue which is deposited in their epithelial layer stratum and which is derived from the lymphatic tissue of the pharyngeal ring.

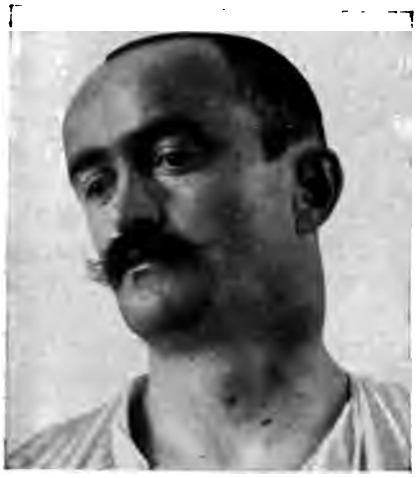


FIG. 108.—Deep cyst of the branchial cleft. (Deep dermoid of neck.)

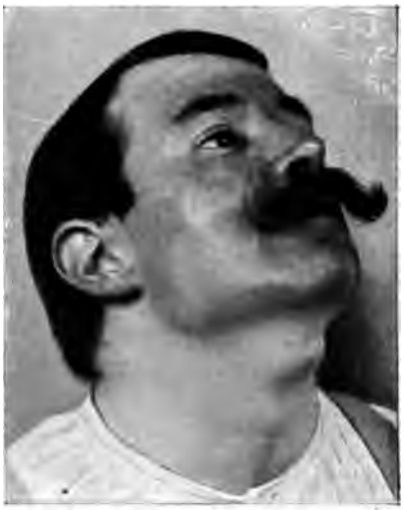


FIG. 109.—Sebaceous cyst, in exact position of median cyst of neck, but more superficial.

The diagnosis of the so-called branchial cleft cysts is not usually difficult, even when the tumour is completely closed in. But in most cases there is a small depression in the skin over the tumour, which renders it impossible to mistake the condition, and facilitates the diagnosis for beginners. The patient probably informs us that a whitish fluid exudes from it occasionally, whereupon the "gland" vanishes, only to reappear again in a short time. Sometimes this evacuation is preceded by an inflammatory attack, and then the exudation is more or less purulent. The picture is complete if we find a small scar in the vicinity of the sinus. We must not be misled into diagnosing tubercular glands, because we find the condition on both sides. Branchial cleft cysts and sinuses may occur on both sides, symmetrically arranged.

Sebaceous cysts situated *in* the skin, in favourite situations of the neck, must not be mistaken for branchial cleft cysts.

(3) SOLID TUMOURS OF THE NECK.

We now proceed to deal with the solid tumours of the neck. As we have already mentioned, a **lipoma** is on the borderland between the fluid and solid tumours, and we should think of it when palpation leaves us in doubt as to which variety is in question. We shall not here refer to the easily recognizable superficial lipoma, which we shall discuss in connection with tumours at the back of the neck—the usual site of this tumour. The *deep sub-fascial* lipoma occurs, but rarely, to add to diagnostic difficulties ; and although it is well defined, its processes permeate between the various organs in the neck.

The questions which centre around most solid tumours of the neck are quite of a different character. These tumours are usually distinctly firm in consistence, if not hard, and we have, therefore, to decide between fibroma, sarcoma, and carcinoma.

Having recognized that the tumour is primary, the question of its innocence or malignancy arises. If the growth has taken years to develop, and still remains movable, it must be regarded clinically as innocent, though it should not be forgotten that a **fibroma of the neck** may be on the borderland between innocence and malignancy, and that it may spontaneously become malignant after many years' existence.

It is only in a very few definite situations that any question of differential diagnosis can arise as between a fibroma and some other innocent tumour. It may, however, occur in the neighbourhood of the submaxillary or the parotid gland, including the region behind the ear. For the purpose of making a general survey, we propose to discuss separately the various sections of the side of the neck.

(a) Submaxillary Region.

Chronic inflammatory processes are occasionally observed in the *submaxillary gland,* leading to its enlargement through increase of the connective tissue, while the glandular tissue itself diminishes. The diagnosis generally points to new growth, and the comparatively rapid growth usually suggests malignancy. It is quite easy to demonstrate by palpation that the growth is connected with the submaxillary gland.

As it is not always possible to distinguish this condition accurately from a real tumour without the microscope, and as the loss of one submaxillary gland is not a very serious matter, the course will usually be adopted of removing the structure.

This chronic inflammation of a *single* submaxillary gland must not be confused with the symmetrical chronic inflammation of *all* the salivary and lachrymal glands, known as *Mikulicz's disease.* In some cases this enlargement is merely one manifestation of leukæmia or lymphadenoma. Tuberculosis of the submaxillary gland is so rare, that it can only be recognized by the microscope, unless the diagnosis is suggested by a chronic abscess.

Omitting rarities, the only innocent tumour amongst those which are genuine growths, which we need consider, is the so-called **mixed tumour.** A movable hard tumour which has slowly grown in the submaxillary region is a fibroma of the neck, if we can feel the salivary gland quite separate from it, on making bi-manual palpation, externally and in the mouth. If the gland cannot be demonstrated as distinct from the tumour, and if it has a smooth surface, it is suspicious of chronic fibroid inflammation of the salivary gland. If the new growth is perfectly movable, but has a strikingly nodulated surface and has existed for years, we should regard it as a so-called mixed tumour. If we learn that the tumour has been noticed for many years, but that the rate of growth has increased rapidly within the last few months, we must assume that it is a mixed tumour which has become secondarily *malignant*, a not infrequent event. In these cases the growth soon loses its mobility, and at the operation it cannot be shelled out as easily as an innocent mixed tumour. Primary malignant tumours of the gland are very rare, and we will discuss them in common with those of the parotid.

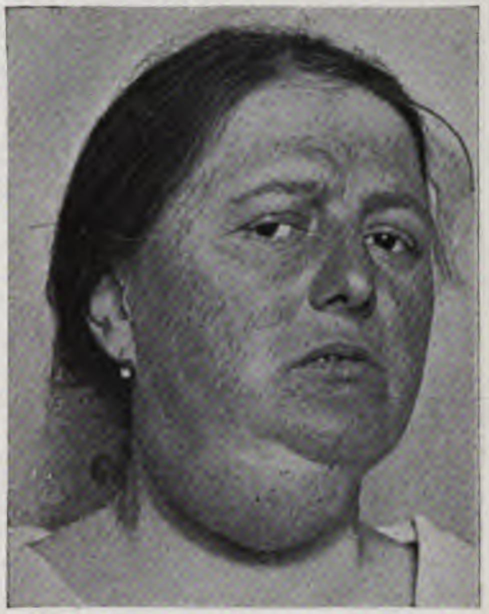


FIG. 110.—Mixed tumour of submaxillary gland.

On examining these mixed tumours a little more closely, we shall see on section that they are partly fibrous, partly gelatinous and partly like soft cartilage. Histologically they consist of a richly cellular tissue in which there are regularly arranged strands of cells, called by some observers epithelial cells, and by others, with equal confidence, endothelial cells, but the microscope affords no definite evidence of their malignancy. In addition there are numerous islets of cartilage, and areas which have undergone mucoid degeneration.

(b) Parotid Region.

The considerations referring to the submaxillary region hold good for the parotid region, but here the differential diagnosis is more limited in one respect, although more extensive in another. Fibroma does not occur and chronic inflammatory processes which look like growths have not been observed in the parotid, with the exception of the diffuse swellings, which have been described in syphilis. But, on the other hand, there is the possibility of confusing **tubercular glands** with a new growth. It is easy to avoid this confusion in the submaxillary region, because the tubercular glands are multiple and can be felt separately. In the parotid region, however, there are small lymphatic glands within the capsule of the salivary gland, and when these become tubercular, they gradually lift up the capsule, still being confined within its limits. It may, therefore, be quite impossible to feel the individual glands as separate structures, because they constitute as it were a common growth within the capsule. The presence of other glands in other situations of the neck might of course lead us to think of tubercle.

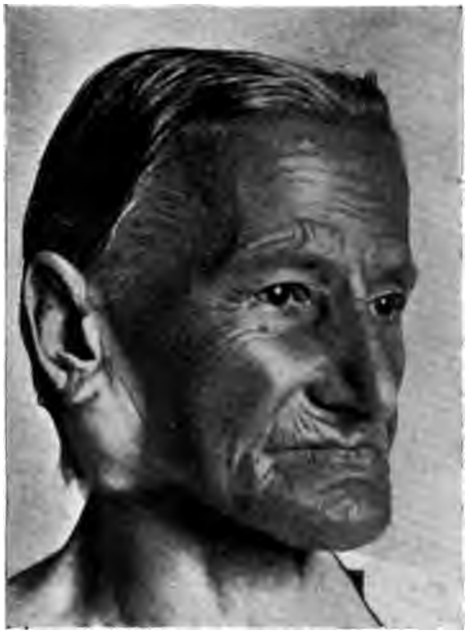


FIG. 111.—Mixed tumour of the parotid, raising up the lobe of ear.

A female of healthy antecedents, aged 30, consulted her family doctor because of a swelling, elastic in consistence, which was gradually making its appearance in the parotid region. Lower down in the neck there were a few soft glands, apparently deeply situated, but not adherent to their surroundings. The facial nerve was unaffected and there was no pain. Everything pointed to tubercular glands rather than to a growth. But the shape of the tumour, which corresponded to that of the parotid gland, including even its posterior process, behind the lobule of the ear, looked startling and made one think of a new growth. Nevertheless the operation showed that the case was one of tubercular glands; they were as if moulded within the parotid capsule, and the parotid tissue itself was entirely displaced into the deeper parts.

Tuberculosis of the glandular substance itself, which is a rare condition, must be distinguished from tubercle of the lymphatic glands over the parotid. If the patient is not tubercular and there is no chronic abscess present to give a clue, it is hardly possible to make the diagnosis. At any rate, we are not able to differentiate it clinically from tubercle of the lymphatic glands over the parotid.

If we have excluded tubercular disease, the innocent tumours which remain are the mixed tumour, described in connection with submaxillary gland, and pure enchondromata, which are rare. The **mixed tumours** of the parotid are so typical of new growths, that no mistake can be made in their diagnosis, even if they do not present those grotesque shapes which used formerly to be seen. Every movable irregular nodular tumour of the parotid region must, at first, be assumed to be a mixed tumour, but the question at once arises, whether it is still innocent. The absence of facial paralysis and of radiating pains in addition to its free mobility should be conclusive in this respect. Pure **enchondromata**, apart from the pre-auricular skin appendages which contain cartilage, are of much rarer occurrence.

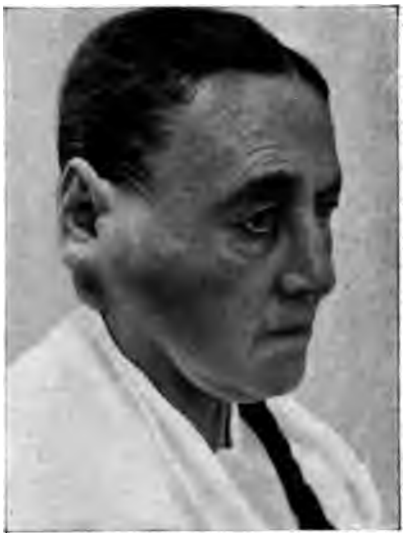


FIG. 112.—Cancer of the parotid. The lobule of the ear is pushed forwards by the posterior process of the gland.

We now come to the primary **malignant tumours** of the submaxillary and parotid region, whose chief symptoms—adhesion to the surrounding parts, especially to nerves—we have already noted.

We conclude that a case is one of *primary* sarcoma, or carcinoma of the affected salivary gland, from the absence of any stage of long duration which is characteristic of an innocent tumour, and which could hardly be overlooked by the patient. Can we go beyond this and distinguish between carcinoma and sarcoma? As a rule we cannot, because even the histological picture of salivary gland tumours is variously interpreted. Experience, however, teaches us that cancer is more frequent in the salivary glands than is sarcoma.

It should be added that cancer does not always exist in the form of a "tumour." The parotid is sometimes affected by a *scirrhous cancer,* just as the breast, in which condition the skin is puckered rather than swollen. Such new growths manifest themselves early by means of radiating pains, especially towards the head, and also by paralysis, or paresis of some of the facial twigs, if not of the whole nerve. If such symptoms are present we must not neglect making a thorough examination of the parotid region, although there may be no tumour apparent.

(c) Side of Neck.

We now come to the side of the neck in the more limited sense. As mentioned above, the *innocent* tumours in this region are mostly *fibromata*, or *neuro-fibromata ;* the primary malignant tumours are, with two exceptions, *sarcomata*.

Cancer of the œsophagus and trachea are not included here, because they are fatal before they give rise to any growth externally. Cancer of the cervical portion of the gullet may at times be palpable from the outside, but the difficulty in swallowing is so prominent a feature that the site of the disease is always perfectly clear.

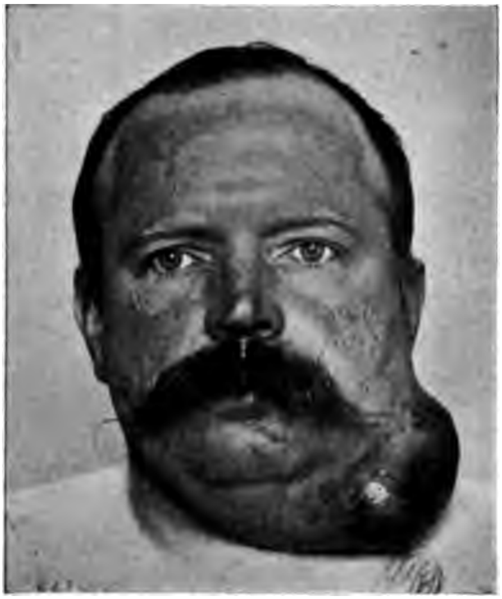


FIG. 113.—Sarcoma of side of neck.

Fibromata and **neuro-fibromata** occur as slowly growing, firm, movable, spindle - shaped, or oval tumours, over which the skin is freely movable. If their origin is deep—from the sympathetic, from the pre - vertebral connective tissue, or from the vertebral column itself—their position is more or less retro-pharyngeal and they cause correspondingly early symptoms of dysphagia and even of dyspnœa. These tumours are frequently confused with fibromata growing from the base of the skull and with retro-pharyngeal abscess. The same applies to **fibro-sarcomata**, except that these grow more rapidly and are less movable. Even with the microscope their distinction from fibromata on the one hand and sarcomata on the other is very difficult.

Neuro-fibromata have a special tendency to become sarcomatous. Often they are only one indication of a general neuro-fibromatosis known as " Recklinghausen's disease." Congenital psychical abnormalities are often associated with this disease, viz., imbecility, infantilism psycho-neuroses. The fibromatosis may encroach on the central organs and give rise to the most varied nerve symptoms.

Sarcoma of the neck may originate in any of the connective tissue structures of this region, *e.g.*, fascia, periosteum, muscular connective tissue, nerves, &c. Its favourite seat is, however, in the lymphatic glands, even leaving out of consideration their enlargement in malignant lymphoma.

In connection with *lymphatic gland sarcoma* it is necessary to distinguish between "*lympho-sarcomata*" and "*sarcomata of the lymphatic glands.*" The former are sarcomatous proliferations of the lymphatic tissue; the latter are sarcomata of the supporting tissue. The former are, therefore, round small-celled sarcomata, sometimes with an interlacing reticulum; the latter are mainly spindle-celled sarcomata. The difference is interesting from the standpoint of pathological anatomy, but it would be too much to expect that the difference should be detected from clinical signs. If the tumour is definitely soft and the growth is infiltrating, we should suspect "lympho-sarcoma"; if it is definitely hard it should be regarded as a "sarcoma of the lymphatic glands."

"*Malignant lymphoma,*" referred to above, must not be confused with these sarcomata. The former is a constitutional disease, whereas *lympho-sarcoma* and *sarcoma of the lymphatic glands* are tumours of individual glands.

Vascular sheath sarcoma formerly constituted a separate variety. It is undoubted that sarcomata may grow from the sheaths of large vessels, but most of the cases thus described were really sarcomata of the lymphatic glands. We must be reconciled to the difficulty of determining clinically their point of origin, especially as it is usually impossible to do so, even at the operation.

In every case of primary malignant growth of the neck the question, whether it is one of the rare primary forms of **carcinoma,** eventually arises.

Tumours have often been found in the upper part of the side of the neck, which have been demonstrated by histological examination to be cancers, with stratified pavement epithelium and for which no primary growth could be found. Their origin must therefore be ascribed, as Volkmann suggests, to congenitally displaced epithelium of a branchial cleft, or to some persistent epithelium of this rudiment. If, in the position indicated, and in a man of middle age, there be a tumour with the signs of a malignant growth, especially if there are severe pains radiating towards the head and back of the neck, the case is in all probabilty a **branchiogenous carcinoma.**

But an absolutely accurate diagnosis can only be established at the autopsy. A small primary cancer concealed on the posterior surface of the larynx cannot possibly be detected by examination, and this may be the real explanation of a so-called "branchiogenous cancer"—only to be discovered post-mortem. Nevertheless branchiogenous cancers do occur and in some few cases it has been possible actually to demonstrate the transition of a congenital cyst, situated at the side of the neck, into a carcinoma. Malignant growths of the parathyroids may also be regarded as branchiogenous carcinomata in the widest sense of the term, as also the much rarer growths which arise from the post-branchial bodies, the so-called lateral thyroid gland rudiments.

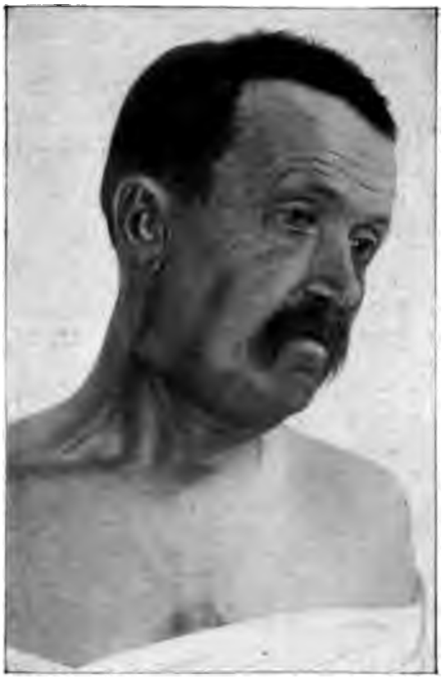


Fig. 114.—Branchiogenous carcinoma.

On the other hand, cancer of the accessory thyroids are not related to the branchial apparatus but to the thyreo-glossal duct. An accurate diagnosis of this growth is impossible without the microscope.

There is yet another tumour, worth bearing in mind, which occurs in the same situation as the usual branchiogenous squamous epithelial cancer. It is neither a carcinoma nor a sarcoma, but a structure, *sui generis,* just like a hypernephroma. It is a new growth arising from the **carotid body,** found in both sexes and at very various ages. It is definitely encapsuled, and its growth extends over many years, thus exhibiting a comparatively innocent character. In this way, it shows a clinical differentiation from a branchial cancer. Vessel walls are constantly observed to grow through, and local recurrence frequently occurs. It is usually soft or elastic in consistence, and its close connection with the carotid artery imparts to it a pulsatile movement.

Microscopically this tumour is similar to a normal carotid body. It is composed of a tissue made up of polygonal epithelioid cells, like the main structure, and is traversed by blood spaces invested by endothelium, after the manner of a sponge.

(d) Supra-clavicular Region.

The genuinely primary tumours of this region are the rare deep lipomata as well as fibromata and sarcomata. **Cervical ribs** often give rise to errors of diagnosis, and we shall therefore now proceed to discuss them.

A cervical rib is a small hard structure which can be felt in the supra-clavicular fossa. Its existence may give rise to no subjective symptoms, but on the other hand it may cause neuralgia or paresis of the nerves of the arm, and exceptionally it may produce circulatory disturbances in the subclavian artery.

Sometimes these symptoms of pressure on the nerve plexus or on the artery only supervene after some definite cause. Thus I have seen, in a soldier with a cervical rib, complete obliteration of the radial pulse from the effect of the leather strap of his knapsack.

The subclavian artery either runs over the rib, or in front of it. The abnormality is usually bilateral, but more pronounced on one side than on the other. Vary rarely two ribs are found on the same side, with the subclavain artery running between them.

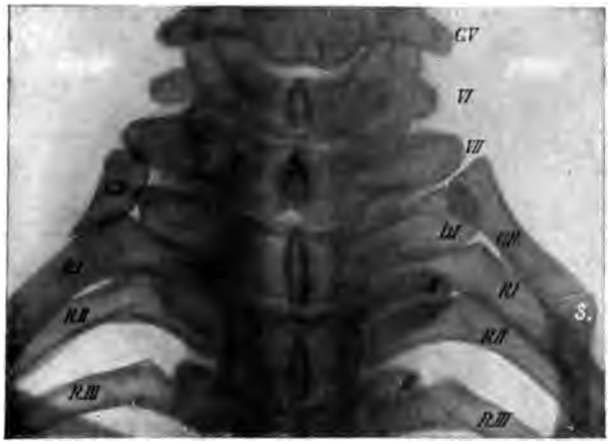


Fig. 115.—Bilateral cervical ribs. On the right, a small rudiment *C R'* attached to the first rib. On the left, a larger rib which is continued to the sternum by means of a clasp *S*. *R I—III* indicate 1st, 2nd, and 3rd ribs. *C V—VII*, *D I—III* are placed on transverse processes of cervical and dorsal vertebræ.

If the medical attendant does not bear in mind the possibility of a cervical rib and is misled by the hardness of the structure, he will diagnose a malignant growth; if he is struck by the pulsation of the subclavian artery, which is pushed forwards, he will regard the case as an aneurism, as the following instance illustrates.

A middle-aged male consulted his doctor in reference to throat trouble. The latter found, after a conscientious examination, a small pulsating tumour in one of the supra-clavicular fossæ, and he thought of a subclavian aneurism. But a closer investigation revealed a small hard structure over which the subclavian artery ran, but the artery was not enlarged—hence the condition was one of *cervical rib*. The diagnosis was clearly confirmed by a skiagram (fig. 115). The throat trouble was due to chronic pharyngitis.

If the hard resistance were strikingly great, one would think of a *chondroma* or *osteoma* starting from a cervical rib. If the pulsating structure near or over the cervical rib were larger than the circumference of a normal subclavian, one would entertain the possibility of an aneurism caused by a cervical rib.

C.—THE BACK OF THE NECK.

We shall conclude the discussion of tumours of the neck with the new growths of the posterior cervical region.

A median, cystic, soft, elastic or fluctuating tumour is usually a **meningocele** or **meningo-encephalocele** (fig. 21); rarely a **dermoid.** The contents of the first two varieties are displaceable on pressure, and vary in tension according to variations in cerebral pressure. They are usually found in children only, because the patients generally succumb unless successfully operated on. But dermoids are found at a later age also, as well as superficial **sebaceous cysts.**

Apart from these, the most frequent tumours in this region are **lipomata.** If the structure is lobulated, soft in consistence, single, and laterally situated (fig. 116), it is an ordinary *encapsuled lipoma,* which can be shelled out with the greatest ease. If, on the other hand, there are two tumours, symmetrically placed near the middle line, which are not definitely lobulated but rather nodular and hard, with no tendency to become pendulous, the diagnosis is *symmetrical posterior cervical lipoma,* a condition which occurs especially among disciples of Bacchus. Frequently these tumours are accompanied by another pair, situated lower down (fig. 117).

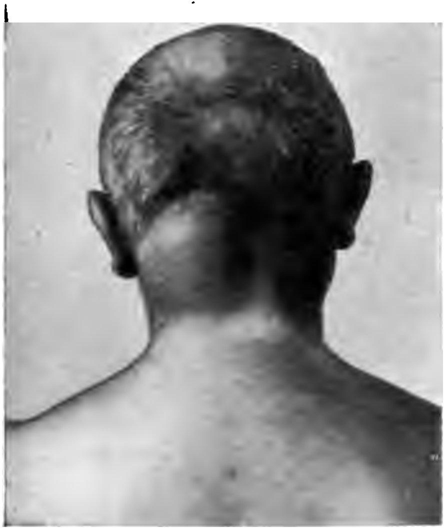


FIG. 116.—Unilateral lipoma of back of neck.

Beginners should be warned that the removal of these tumours should not be lightly undertaken. They dip in between the interstices of the muscles, and have extensive adhesions, so that the operation is very troublesome and apt to be attended by severe hæmorrhage.

Peri-glandular lipoma is a third form of lipoma, which occurs not only at the back of the neck, but also in other situations of the neck and body generally, as circumscribed accumlations of fat around lymphatic glands (fig. 118).

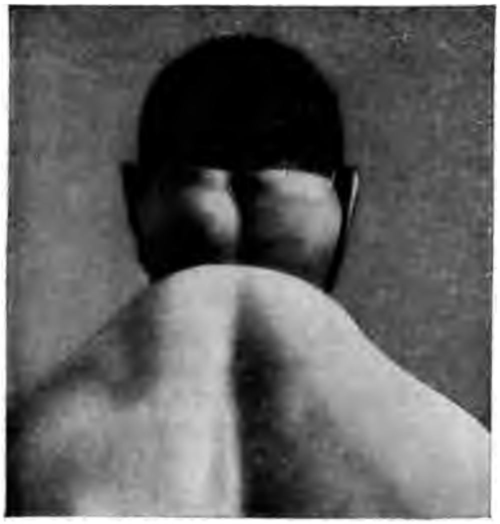


FIG. 117.—Symmetrical lipoma at back of neck. (From the Surgical Clinique at Berne.)

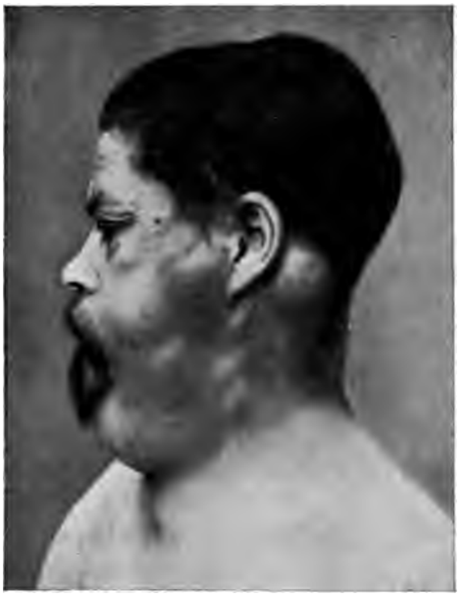


FIG. 118.—Peri-glandular lipoma. (From the Surgical Clinic at Berne.)

Diffuse lipoma of the neck, as described by Madelung, must be distinguished from the ordinary symmetrical lipoma of the back of the neck. The whole neck is surrounded by a fatty mass, as by a collar, out of which the head appears to emerge.

Probably the two latter forms of lipoma have some connection with *adiposis dolorosa* or *Dercum's disease*. This disease is recognized by the distribution of nodular or diffuse masses of fat in different parts of the body, associated with severe pain on pressure, neuralgia, and other sensory disturbances. Certain clinical and anatomical findings suggest a connection between this disease and functional disturbances of the thyroid or pituitary gland (see p. 26).

Hard tumours, mostly situated at the side of the posterior cervical region, are either **fibromata** or **sarcomata.** They may arise from the aponeurotic connective tissue, more rarely from the vertebral spine, but sometimes even from the skin. The rapidity of its growth, its adhesions, its consistence and the condition of the super-jacent skin will indicate whether the tumour is more fibromatous or more sarcomatous in nature, or whether it is a pure sarcoma.

A very rare case is recorded by Dower. This was a fibroma of the dura mater with a slender peduncle, which made its way between the second and third cervical vertebræ, and formed an orange-sized tumour at the side of the neck. The main feature of the case was the compression of the spinal cord. A very similar case was successfully operated on by Wilms.

CHAPTER XXV.

ABNORMAL POSTURES OF THE HEAD.

It is said that a politician once felt much encouraged during the delivery of a long speech by the remarkably attentive posture of his immediate neighbour in the audience. He thanked the listener after the speech, but the latter, not quite appreciating the gratitude, said he had a stiff neck. Obviously the speaker was not a medical practitioner. *We* infer from a stiff neck something quite different to special attentiveness.

We must first note whether the patient carefully avoids any movement of the head for fear of pain, or whether, although he holds the head in an abnormal position, he is able to move it partially without pain.

A.—PAINFUL RIGIDITY OF THE NECK.

Every painful condition about the neck has the effect, on the cervical vertebræ, which we call muscular fixation when it occurs in other joints. A boil at the back of the neck suffices to produce this effect. The nature of the case is evident as soon as we see the patient coming with a compress on his neck or a plaster applied. There is no need for the patient to state his case; but the underlying cause of the condition may be difficult to ascertain. If the muscles on both sides are equally tense and the head is held in the middle line, we may assume that the lesion which excites the pain is centrally situated, whereas if the head is held asymmetrically the lesion is on one side. We shall discuss each variety separately, because the considerations we shall advance proceed from the outset, in different directions.

(1) SYMMETRICAL FORMS.

As the muscular causes which may produce rigidity are generally unilateral and therefore result in *wry neck*, we should at once think of a median structure, *the cervical spine*, when the rigidity of the head is in a *straight* posture. But even then, our conception of the case will vary with the sudden or gradual character of the onset.

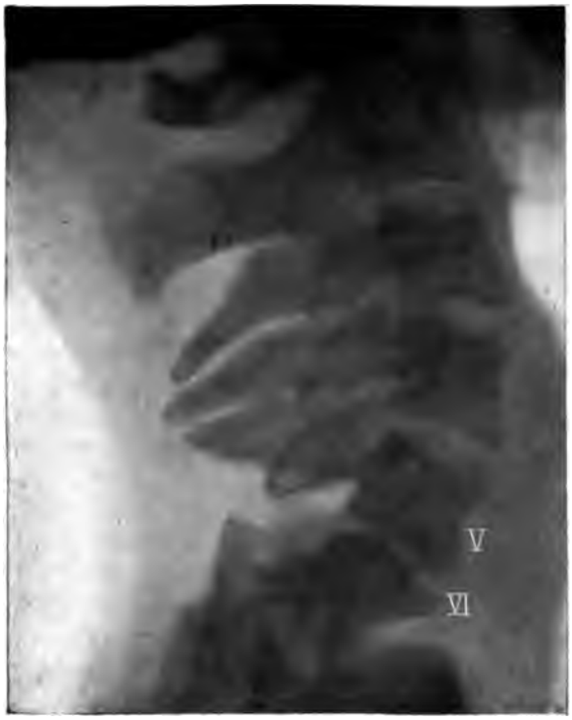


FIG. 119.—Complete forward dislocation of the 5th cervical vertebra on the 6th. Compare fig. 122.

(a) Rigidity with Sudden Onset.

If the rigidity has come on suddenly, we first enquire about an **injury.** In cases of severe injury to the cervical vertebræ, the patient supplies his own indications thereof.

In bilateral or **complete dislocation,** or in fracture-dislocation, with the dislocation still persistent, the profile shows significant displacement of the head forwards, usually associated with flexion. The dislocation always occurs in a manner which causes the upper vertebra to project beyond the lower, and which, with few exceptions, brings about slight flexion at the same time.

We must recall the actions of the various parts of the cervical spine in order to test the functions of the neck. The movement of nodding occurs between the occiput and atlas; the movement of rotation between the atlas and axis, and lower down the chief movement is that of bending the whole neck backwards and forwards. It should be remembered that the various joints of the neck are able, to a great extent, to replace one another.

On palpation an unusual space is to be noted between two spinous processes. The spinous process of the dislocated vertebra is also displaced upwards, touching the spine of the next highest vertebra (fig. 122).

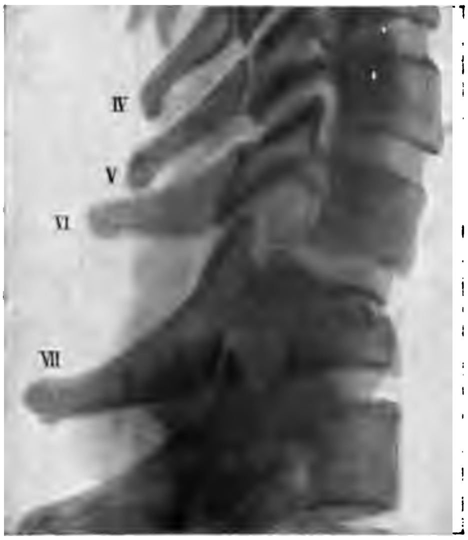


FIG. 120.—Fracture dislocation beween the 5th and 6th vertebræ, fixed in the position of subluxation. (*Post-mortem* preparation.)

In lean subjects the spinous process of the axis can be distinctly felt, but those of the third and fourth vertebræ are indistinct. The spine of the fifth vertebra is again distinct, and those of the sixth and seventh are very clearly felt.

The demonstration of displacement of the lateral portion of the vertebra would be of value in diagnosis, but this is very difficult. On the other hand, a widening of the neck as seen in profile, in addition to bending of the head, is very significant. The displacement which is most common concerns the fifth vertebra, in relation to the sixth, wherein the spinal cord sometimes escapes injury. Complete dislocations higher up are nearly always fatal. But should such a case survive, the diagnosis could be established by feeling through the pharynx and by external palpation of the side of the neck. But sometimes the displacement is very slight in compression fractures (fig. 120), and it is quite impossible to decide upon the nature of the injury merely by means of palpation. A diagnosis can only then be made by X-rays. These are the cases which form the intermediate stage towards **contusion** and **sprain.**

There is no change in the shape of the cervical spine in these latter cases. Spine and lateral processes are in correct position. It is only the active movements which are deranged, and this is quite relative. It is caused, as previously explained, by muscular fixation induced by the pain. Careful and slow manipulation will not only allow of the performance of all passive movements, but will also permit of their active performance. Theoretically there should be no pain in a case of sprain when pressure is made in the long axis, but it is sometimes present because, as a consequence of the normal curvature of the cervical spine, every thrust causes an increase of dorsiflexion and therewith some tearing of the ligaments. If such pressure is very painful it suggests contusion of an intervertebral disc, or even a fracture unaccompanied by dislocation.

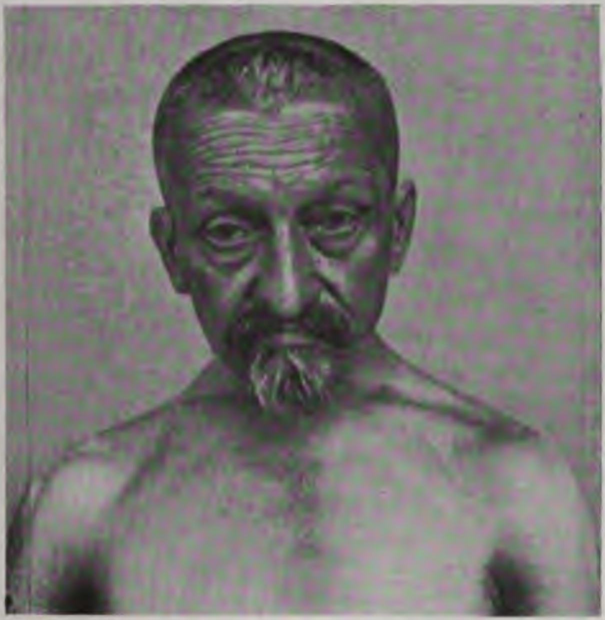


FIG. 121.—Complete forward dislocation of the 5th cervical spine on to the 6th.

On the other hand, the pain caused by pressure in the long axis in cases of severe contusion and compression fracture, is sometimes less than might *a priori* be anticipated.

Pressure on the spinous process may also be painful in cases of sprain, because it may produce slight displacement and tearing of the injured ligaments.

The *site* of the sprain may be inferred from the character of the disturbed functions and from the position of the pain. If the nodding movement is affected, but the neck can still be bent, the injury exists at the occipito-atlantal joint. If there is difficulty in turning the head the sprain is probably between the atlas and axis. If the power of bending backwards and forwards is involved the sprain is more deeply situated. Very careful observation is required to distinguish between nodding movements and bending of the neck, but this differentiation can be made out quite accurately. The existence of damage to the spinal roots (neuralgias) is also of value for the diagnosis.

The foregoing remarks require some amplification. There are some injuries of the first two cervical vertebræ which cause no striking alteration of posture, but produce very severe pain on movement and a corresponding muscular fixation. Fractures of the first two vertebræ are immediately fatal if combined with definite dislocation, but if not so complicated they may recover, unless some unforeseen movement or careless examination causes a dislocation as a supplementary injury. A case of this kind is recorded wherein the patient died suddenly as the nurse was helping to sit him up. These patients are constantly holding their head with their hands, because they feel very uncertain about its stability. The fracture is either in the arch of the atlas or axis, or in the odontoid process of the latter. A fairly accurate diagnosis can only be made in the former condition, if by

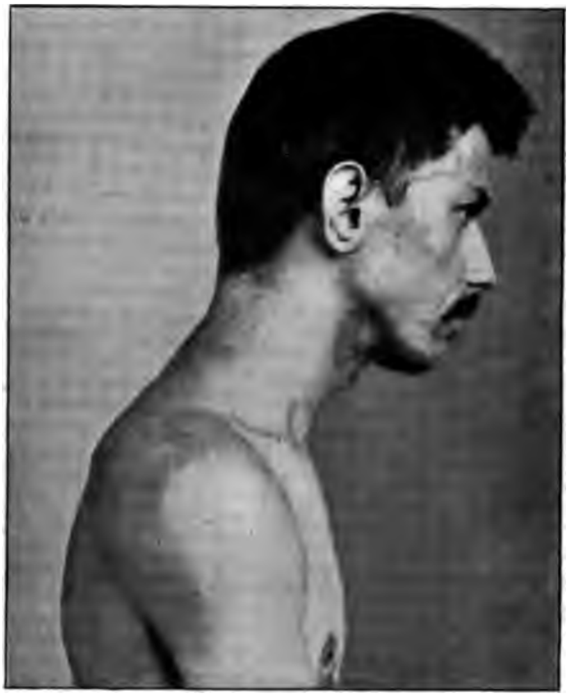


FIG. 122.—Complete forward dislocation of 5th cervical vertebra. Compare with fig. 119.

examination through the mouth of the palpable portion of the atlas it is possible to detect any displacement or abnormal mobility. No other diagnostic manipulation ought to be undertaken if there be any suspicion of such a severe injury. But it is permissible to take an X-ray picture (from the side and from the front with the mouth open) as long as the process can be carried out with the necessary care.

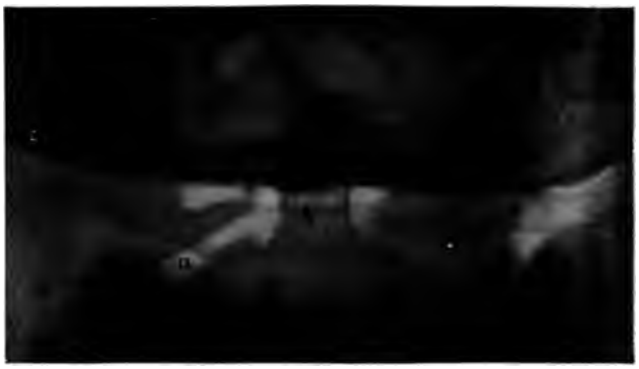


FIG. 123.—Normal vertebra taken with mouth widely open. a, Joint between atlas and axis; b, odontoid process of axis.

FIG. 124.—Fracture of odontoid process, taken through the mouth. The two articular surfaces between atlas and axis are united by line of fracture.

Fractures of the odontoid process occur more frequently than is supposed. They can be clearly shown on a skiagram, and may be suspected clinically from the instability of the head and the tenderness of the vertebra on pressure (figs. 123 and 124).

It is not very easy to distinguish partial fractures of the lower cervical vertebræ without displacement, from sprains. Detachment from the articular process with incomplete rotation—dislocation, is an example of such an injury. A long continuance of symptoms, assumed to be due to a sprain, is strongly suggestive of this lesion, but a decision can only be arrived at by a skiagram.

A patient in whom sudden rigidity of the neck comes on, is not always aware of an antecedent injury. "**Torticollis**" is looked upon very much like "lumbago," and there is always a tendency to discover a chill as its cause.

But the real cause may be a slight twist just as in many cases of lumbago. It is brought about by some fortuitous movement of the neck, in which there has been neglect to fix the individual vertebræ in the necessary manner by appropriate muscular action. Sometimes pains radiating towards the shoulders occur, as in the case of the more severe sprains. But none of the spinous processes are definitely painful on pressure, nor is pressure along the axis of the spine painful. Those who have themselves suffered from this kind of sprain are best able to appreciate its true nature.

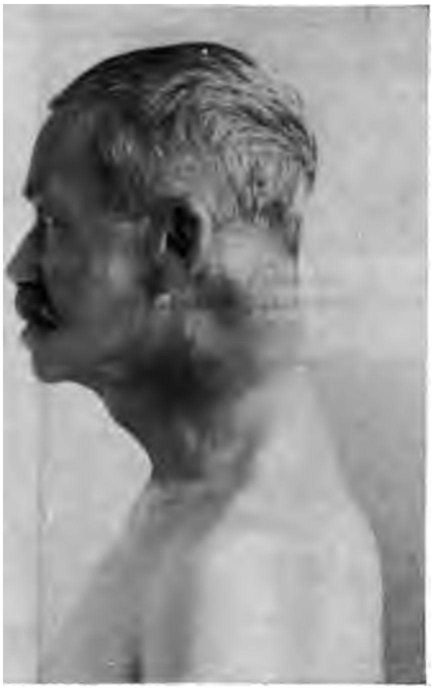


FIG. 125.—Caries of 5th and 6th cervical vertebræ. Head displaced slightly forwards. Neck abnormally wide in profile.

In some cases wherein the head is held fixed in a symmetrical position the cause is a slightly acute cervical adenitis due to sore throat.

If there has been no injury and the symptoms of rigidity are accompanied by rigors and fever, we must think of the possibility of **osteo-myelitis of the spine.** Active movements in the adjacent vertebral joints are in abeyance, there is pronounced pain on pressure over the spine and lateral portions, and if the disease is situated high up, pressure through the pharynx demonstrates pain on the anterior surface of the vertebra. Pressure along the axis of the spine is painful, corresponding to the extent of the inflammation. The diagnosis would be confirmed if the history revealed any recent acute inflammatory disease which might serve as the primary focus. Further confirmation is afforded by the course of the disease, in which nerve root and spinal cord symptoms supervene, and unfortunately by the almost invariably fatal termination.

(b) Rigidity with Gradual Onset.

If the rigidity has come on *gradually* the cause is usually tuberculosis of the vertebræ (fig. 125), or more rarely a new growth. The examination must be conducted most carefully, because any excessive diagnostic zeal may be rewarded by the breaking off of the odontoid process of the axis—an accident which has actually occurred. We ascertain the extent of active movements, the amount of pain on pressure over the long axis of the spine, and on the spinous processes, and finally we test sensitiveness to pressure through the pharynx. We diagnose the site of the lesion, just as we do the site of any injury by means of the disturbance of movement, the change in shape of the spine, and by the situation of the severest pain on pressure. Sharply limited neuralgias often facilitate the local diagnosis. We should also look for burrowing abscesses, and test the patellar reflexes in order not to overlook the commencement of some pressure on the spinal cord in the neck.

Further information is given in the chapter on "Inflammatory Diseases of the Spinal Column."

(2) ASYMMETRICAL FORMS.

If the posture of the head is not symmetrical, but is inclined towards one side and turned towards the other, the condition is one of "*wry-neck*." If this has come on *suddenly*, the question of **myositis** should immediately arise. This diagnosis would be borne out, if a muscle, especially a sterno-mastoid, were not only tense but also swollen and painful on pressure. If the malady follows an acute infectious disease or if it is only one manifestation of multiple myositis with high temperature, the condition is really serious, because it may lead to a permanent wry-neck, through subsequent fibrous degeneration of the muscle. Bot if there are no severe symptoms and the swelling of the sterno-mastoid is the solitary morbid symptom, we may regard the case as one of *rheumatic myositis* and give a favourable prognosis.

Many cases which are called rheumatic myositis are really due to *acute adenitis* of a cervical gland lying beneath the sterno-mastoid, and careful examination will often reveal some dental trouble or sore-throat as the primary cause.

If the muscle is not swollen but some injury has preceded the rigidity the case is probably one of a simple *sprain*, which we shall be able to distinguish from a more serious accident by means of the rules previously given. The more serious unilateral injuries in this situation are usually unilateral **dislocations.** The head is held in such a distinctive manner in these cases that the diagnosis presents no difficulty. The head is bent towards the dislocated side and turned towards the uninjured side (fig. 126).

At any rate this is the posture assumed in cases of unilateral dislocation with interlocking of the articular processes when the articular process of the upper vertebra has become displaced to the front of the corresponding process of the lower vertebra (figs. 128 and 130). But if the twist has only proceeded half way, that is to say, that the articular process of the displaced vertebra rests on the edge of the process of the lower vertebra (fig. 129), we should expect, theoretically, that the head would assume a different posture. The head should be inclined towards the uninjured side, so that the injured side would be lengthened and only slightly turned. This variety of dislocation is, however, so unstable, and carries with it so little risk to life that it has never been demonstrated *post mortem.* Only a skiagram could show whether such a picture, as conceived by most writers, represents the truth. We might suspect this form of dislocation, clinically, in a case wherein the amount of turning was insignificant in comparison to the bending, and where the spinous process and lateral portion were only slightly displaced. The much more frequent cases of complete rotation-dislocation with detachment of an articular process can likewise only be diagnosed by a skiagram.

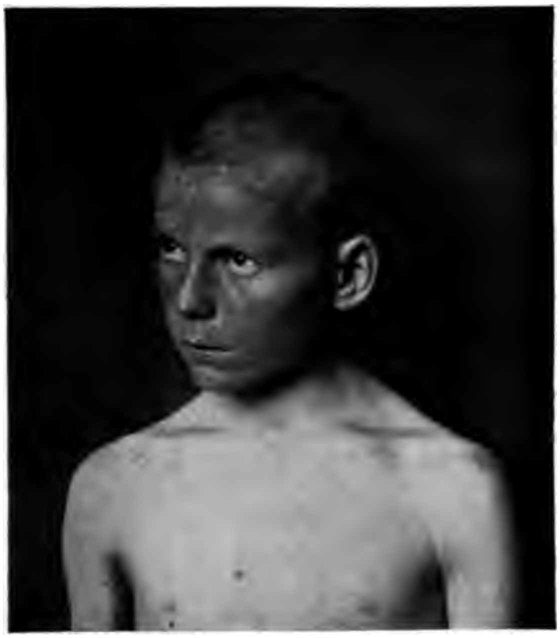


FIG. 126.—Left-sided dislocation between atlas and axis, rotation of head to opposite side.

As in the case of other dislocations, the faulty position in the neck can be increased artificially, whereas considerable resistance is offered to *opposed movement* especially when there is interlocking. Spontaneous pain is often quite trifling, but in recent cases all *movements for the purpose of examination,* as well as pressure on the spine of the dislocated vertebra, are painful. Palpation affords the most feasible proof of dislocation. In the case of the upper vertebræ we must examine through the pharynx and endeavour to make out whether we can detect one side of the vertebra forwardly displaced. Any kind of assymmetry is abnormal. If we are not quite sure about our results, we must examine with the index-finger of the other hand for the

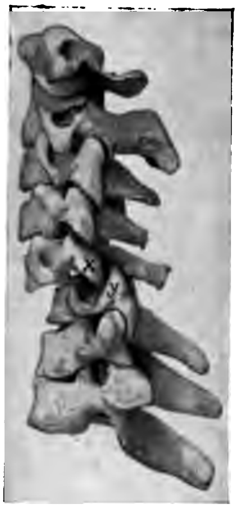


FIG. 127.—Complete dislocation.

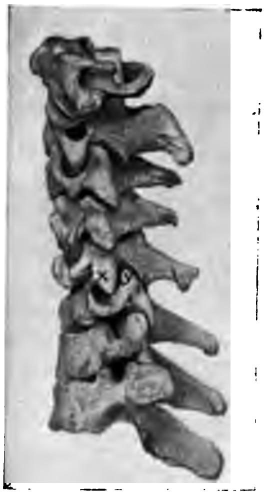


FIG. 128.—One-sided dislocation with interlocking.

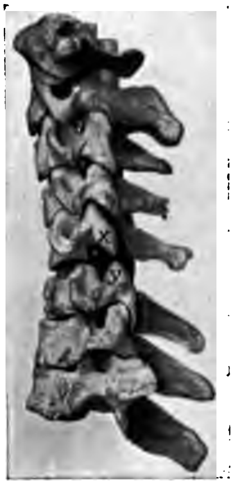


FIG. 129.—One-sided dislocation without interlocking.

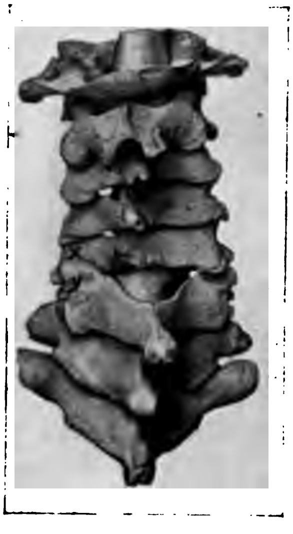


FIG. 130.—One-sided dislocation with interlocking.

purpose of control. In this way it is quite possible to recognize dislocation of the first two vertebræ, and of the third vertebra also if the examining fingers are long enough and the patient's neck not too long.

An examiner with specially long fingers has been able to reach as far as the sixth vetebra in toothless corpses with short necks, but in living persons this is impossible.

On the other hand, no reliable results are yielded by external palpation of the spinous processes and the lateral portions in rotation-dislocation of the lower vertebræ.

Spinal caries and tumours must be mentioned among the causes of painful wry-neck. It the damage is only on one side it may imitate the signs of a one-sided dislocation or rather it may lead to that condition.

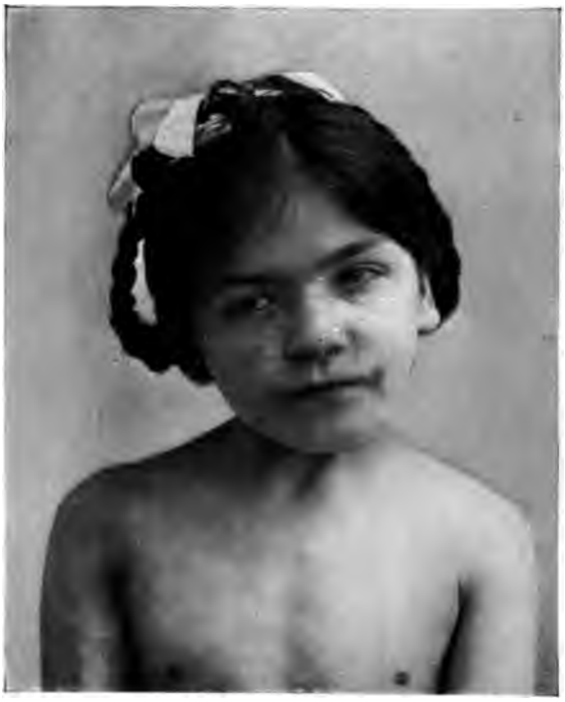


FIG. 131.—Congenital torticollis.

B.—PAINLESS RIGIDITY OF NECK.

It will, of course, be understood that these cases are always chronic. A *symmetrical* rigidity must be due either to some form of painless spinal caries, to an old bilateral dislocation or to a healed compression-fracture. But if the condition is one of *wry-neck* and an old unilateral dislocation can be excluded by palpation, the case must be grouped under the extensive class of so-called **caput obstipum** or **muscular torticollis.**

The causation of this common malady is still a subject of controversy. Stromeyer and many others attribute it to injury of a sterno-mastoid during birth, with subsequent fibrous degeneration and contraction of the muscle. Petersen's view is that the condition is of intra-uterine origin, due to the cramped space. He bases his view on the cases which are undoubtedly congenital and even hereditary. This conception is confirmed by recent observations (Voelker), because it has been shown that the shoulder pressing against the neck *in utero* leads to atrophy of the sterno-mastoid. Kader, however, as a result of definite observations, attributes all wry-necks to the result of *infective myositis,* which comes on *after* birth, but very frequently owing to trauma during birth. According to Mikulicz, this explanation would account for *intra-uterine* shortening of the sterno-mastoid.

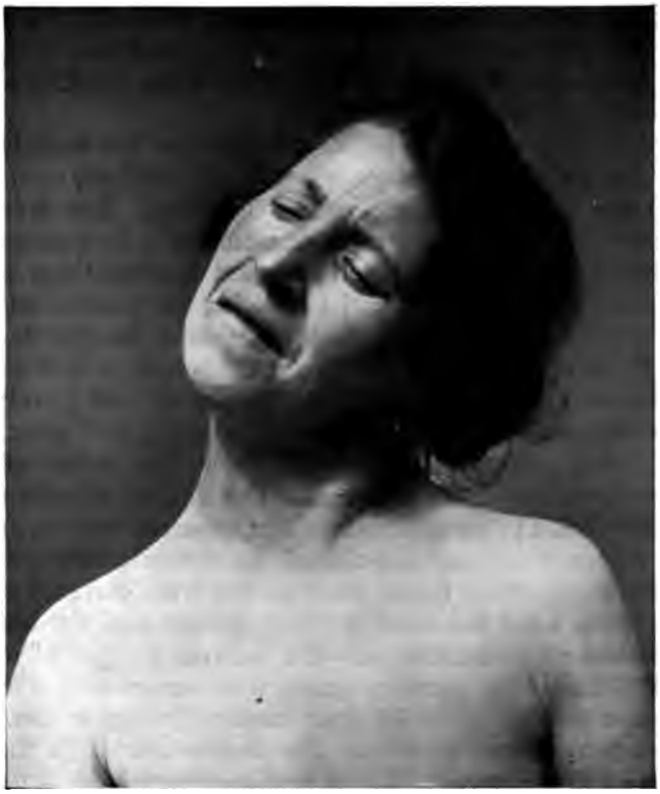


FIG. 132.—Spastic torticollis.

However this may be, the chief factor in diagnosis lies in the circumstance that the malady has appeared during infancy. This explains the fact that the whole skeleton has become adapted to the abnormal posture of the head. The skull is asymmetrical, shortened and widened on the affected side, the spinal column shows cervical scoliosis with a continuation thereof in the dorsal region, the convexity being towards the healthy side. Sometimes there is, in addition to the cervical scoliosis a dorsal scoliosis towards the opposite side and lumbar scoliosis towards the same side as the cervical scoliosis. The one sterno-mastoid in the neck is shortened, resembling a narrow, hard, projecting band, whereas the other is often abnormally well developed. The most striking thing about the posture of the head, especially in children, is the inclination of the neck towards the affected side, with comparatively slight rotation towards the healthy side (fig. 132). As the disease progresses this lateral inclination of the head diminishes but the rotation increases, so that the head may deviate entirely towards the healthy side. The complicated vertebral curvatures also belong to this latter category.

There is, finally, another clinical picture, which essentially belongs to the department of medicine, but which often appeals to surgery when therapeutic measures have failed.

As soon as the patient begins to describe his malady—which, however, is quite unnecessary—his head is suddenly and violently jerked to one side and turned towards the other. The more excited he becomes thereby, and the more anxious he is to impress us with his distressing condition, the more frequent become these spasmodic movements. Sometimes the facial muscles, the muscles of the floor of the mouth and even the shoulder muscles co-operate in these convulsive movements. They are sometimes intermittent and clonic, at other times persistent and tonic, involving not merely isolated muscles, but muscles which work co-ordinatèly, and muscle groups on both sides. For this reason the older designation of "spinal accessory convulsions" is incorrect. The clinical picture represents **spastic torticollis** in its most usual form, wherein one sterno-mastoid and the posterior cervical muscles of the opposite side act together. There are other allied forms such as bi-lateral contractions of the muscles which bend the head, nodding spasms and the contractions of the posterior cervical muscles—the "retrocollis spasm" of English authors. There are some patients who experience the greatest difficulty in putting food in their mouths owing to these spasms. I know a practitioner in whom the disease started in the form of a writer's cramp in the right arm, and as the malady progressed the shoulder muscles participated in the spasmodic movements of the head. No wonder, then, that these patients turn to the surgeon for relief when internal treatment has failed.

It is still uncertain whether the situation of the disease—which must be looked upon as a neurosis—is exclusively in the cerebral cortex or also in the more deeply placed centres of co-ordination. Possibly both are involved. At any rate it is quite certain that the results of operative treatment, division of the muscles which take part in the convulsions cannot be attributed to pure suggestion. It is more probable that the irritable cortical centre is put out of action for a considerable time owing to the absence of centrifugal impulses from the tense muscle, and thus repose is ensured for it.

PART III.

SURGICAL DISEASES OF THE THORAX.

CHAPTER XXVI.

FRACTURES OF THE BONES OF THE THORAX.

It is obvious that the ribs may be fractured by means of severe violence, directly—at the site whereon the force is applied—or indirectly at the site where the curvature is most pronounced. But it is not quite so easy to appreciate that a rib may break when the violence is very slight, or even as a result of muscular contraction only. Such cases, however, presuppose a debilitated osseous system through one cause or another, mostly senility. I knew an old man who fractured a rib through cutting a loaf. Ribs have been broken during labour through muscular contraction, and even while sneezing. After a severe injury, in which several ribs have been broken, it may be quite possible to hear crepitus with each breath, even in an adjoining room, but in the slight cases just mentioned it is often necessary to search for the injury in order to make an accurate diagnosis.

The outstanding symptom is pain felt as each breath is drawn, pain which effectually prevents any deep inspiration. Gaping, sneezing and laughing are particularly painful, and it was, therefore, a thoughtless joke on the part of a medical student who sent a colleague suffering from fractured ribs an amusing newspaper article to cheer him up, the perusal of which brought into play all the muscles required for a hearty laugh. The interference with respiration is not necessarily the result of a fractured rib; it may be due to a hæmatoma in the muscle or beneath the pleura. Both these conditions are, however, very frequent in cases of fractured ribs.

If, on palpating the ribs, we feel or hear crepitus anywhere on deep respiration, there can be no doubt about fracture. The stethoscope may be of assistance in this examination. But sometimes we only find a very painful spot without any signs of abnormal mobility. This may indicate nothing but a contusion. The distinction is made by the possibility or impossibility of eliciting pain indirectly by making pressure and counterpressure on both extremities of the ribs, *i.e.*, by increasing their curvature. Pain produced in this way, remote from the points of pressure, is significant of fracture or, at any rate, of incomplete fracture of a rib.

If this examination elicits pain, both in the back and in the front, it follows that the rib has been broken behind as well as in front, a frequent incident in contusions of the whole thorax. In these cases there is usually concomitant damage to several other ribs.

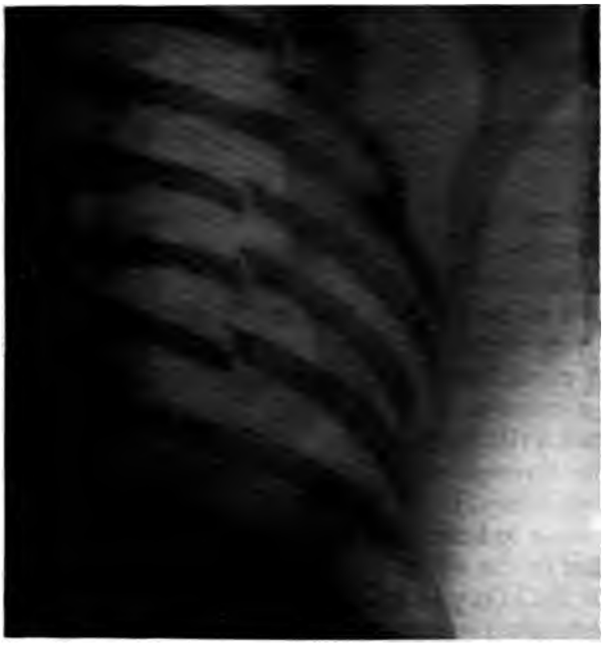


FIG. 133.—Multiple fracture of ribs.

Fracture of the sternum sometimes runs its course quite unnoticed, because the injury is rather of the nature of a fissure than a complete fracture with abnormal movement and displacement. It is impossible to mistake this latter form owing to the superficial position of the sternum.

The following case refers to an unusual variety of dislocation :—

A workman fell against a box which he was holding in front of him, in such a way that the ensiform process hit against its edge. When the patient undressed he noticed a remarkable projection in the epigastrium. The sternum had been transversely split at the level of the fifth rib, and was displaced towards the skin. The rib cartilage was bent forwards and held the displaced fragment so firmly in position that reposition by the open method became necessary (fig. 134).

In cases where the accident is nothing more than a simple transverse fissure, notice is first attracted to the injury by the ecchymosis, which comes on in the course of a few days. As stated elsewhere, these fractures are generally indirect, and accompany fracture of a vertebra. It is, therefore, most important to examine the vertebral column in every case of fractured sternum, and *vice versâ.*

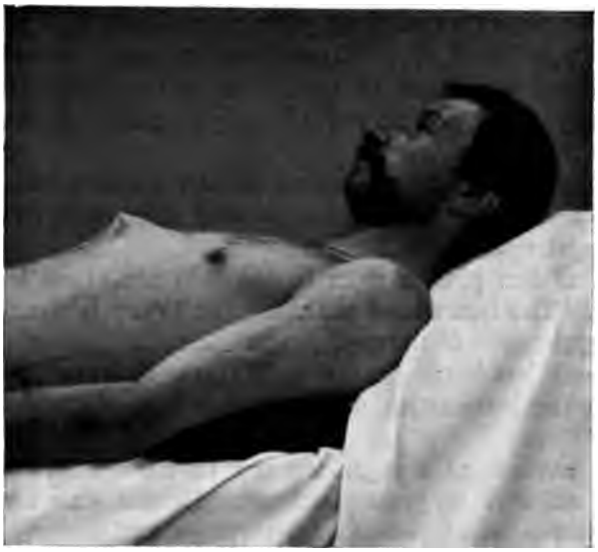


FIG. 134.—Fracture of the sternum with projection of lower fragment externally.

CHAPTER XXVII.

INJURIES OF THE LUNG.

IF a liquid effusion rapidly makes its appearance within the thorax of a patient suffering from **contusion** thereof, or from fractured ribs, we must diagnose *hæmorrhagic effusion,* which may originate either from a ruptured intercostal artery or from the blood-vessels of the lung. We can only be certain of the latter origin if signs of *pneumothorax* supervene upon those of the effusion, or if the patient *coughs up* blood. The injury to the lung becomes quite obvious if air penetrates into the subcutaneous tissue and gives rise to the well known appearance of *cellular emphysema,* which often extends over a very wide area. Death ensues rapidly if a main bronchus has been completely torn away from the lung and opens into the mediastinal cellular tissue and inflates it. The effusion of blood and the pneumothorax must be carefully watched, because their increase, in association with a worse condition of the pulse and respiration, may demand operative interference. Sometimes, however, the initial symptoms of contusion of the lungs are so slight that the injury is overlooked, and it is only diagnosed after consecutive **pneumonia** has arisen.

Pneumonia, following contusion, usually appears within the first four days, a circumstance which is very important in arriving at a medico-legal opinion in doubtful cases.

Injuries of the pleura open to the skin are of no less practical importance. The instrument inflicting the injury is usually a bullet, knife or dagger, even in times of peace.

The thorax has been pierced right through, from axilla to axilla by the domestic broomstick, as recorded by Franke, with subsequent recovery.

Gunshot wounds of the pulmonary tissue are remarkably well tolerated because of its great elasticity. Recent wars have afforded numerous instances of people shot through and through, who kept on marching or riding for hours, and who returned to duty after a brief detention in the field hospital. This applies, at any rate, to injuries inflicted by modern small-calibre bullets with their small surface of attack. The coughing up of blood-stained expectoration, once or twice, may be the sole evidence of injury to the lung. More serious results follow, especially severe hæmothorax and rapid hæmorrhage, from shrapnel bullets or pieces of shell, or when the projectile, on its way through the thoracic wall, has torn up buttons or splinters of rib, or has opened up a large vessel.

Incised and *penetrating wounds* of the thorax cause expectoration of blood, whether the lung is involved or not. A simple hæmothorax is not conclusive of the precise nature of the injury, because it may originate from a wounded intercostal or mammary artery as well as from a pulmonary vessel. A pneumothorax is only significant of an injured lung if the condition continues to increase when the external wound is hermetically sealed.

We must also be careful in diagnosing emphysema in cases of injury to the thorax. Whereas an extensive surgical emphysema points definitely to an injury of the lung, the slighter forms are due to aspiration of air through the external wound if the latter is situated in the vicinity of the armpit, and the patient is frequently lifting and dropping his arm.

In conclusion, a very important rule for the examination and treatment of injuries to the lung. This rule emphasizes the utmost importance of *rest*, because every change in position accelerates the respiration and may cause a fresh hæmorrhage. Diagnostic energy must, therefore, not be pushed too far, and we must be satisfied to move the patient as little as possible.

Although we may be clear about the injury to the lung, we must not forget that it is often accompanied by **secondary injuries**. The diaphragm and the viscera in relation therewith are especially endangered. A stab in the right side often involves, not only the lung but also the diaphragm and liver. We must, therefore, never neglect to examine for hæmorrhage within the abdomen, lest a patient may be bleeding from a wound of the liver while we assume he has only sustained an injury to the lung, not of a dangerous character. The same consideration applies on the left side, to the spleen. The stomach is less frequently involved, because it more easily escapes the knife than the solid viscera. On the other hand a pleural injury may be overlooked when the direction of the inflicted wound runs from below upwards, penetrating the abdomen first. I have seen the bile, in such a case, escaping from the liver, which had been simultaneously injured, not into the abdominal cavity, but into the right pleural space. Injuries to the diaphragm on the left side produce immediate or subsequent *diaphragmatic hernia*. The following case shows that this result is not limited to penetrating injuries like gunshot wounds or stabs.

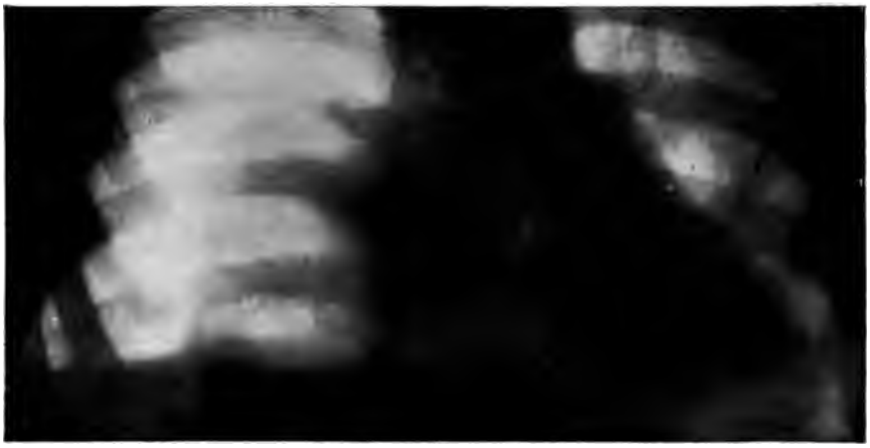


FIG. 135.—Pneumothorax through detachment of right bronchus. Considerable clear area. Contracted lung lying against median shadow.

A workman was brought to hospital in a moribund state after a severe crush of the thorax. The autopsy revealed several broken ribs, with a deep laceration of the left lung caused by the point of one of the rib fragments. The same fragment had pierced the diaphragm, and the wound therein was immediately filled up by a fungiform plug of omentum which projected into the pleural cavity.

Injuries to the lung are sometimes complicated by *wounds of the heart*, which then demand the chief attention, as being of the greatest danger. If the injury involves the *large vessels* in the mediastinum, an autopsy is inevitable. The easily deflected *œsophagus* is least exposed to danger.

CHAPTER XXVIII.

INJURIES OF THE HEART.

TWENTY years ago, death from wounds of the heart seemed quite the obvious thing, and recovery was ascribed to a lucky accident. Suture of the heart has however now been performed some dozens of times and in two-fifths of the cases the result has been successful. But it is necessary that the diagnosis should be made with rapidity and accuracy if surgical interference is to be afforded adequate opportunity.

The *position* and the *character* of the wound are the principal indications which raise the suspicion of injury to the heart. Any wound situated over the cardiac area or its vicinity should excite suspicion, and in this connection it must not be forgotten that the heart may be wounded, in exceptional cases, by stabs or shots from behind. If the wound is not directly over the heart, the direction and the length of the instrument which inflicted it will show whether it is possible that the heart may have been involved.

The *probe* must *never* be employed in examination. The only reason for determining whether the wound is a penetrating one is to ascertain the presence of an injury to any underlying organ, but the probe is unable to give information in this type of case. The probe may actually impinge upon the heart and yet we may be unable to tell whether it is injured or not. Beyond this, there is an element of danger in probing an injured heart, for this procedure has restarted a hæmorrhage which had already ceased. The patient is also unnecessarily exposed to the risk of infection because the superficial parts of the wound are not always aseptic.

If the wound is large, examination may be made with the finger, which will, as a matter of fact, enable a conclusion to be arrived at better than a probe, especially if the finger not only explores *over* the heart but also the actual wound itself, as recommended by Longo. But we should only resort to this not entirely harmless examination when everything is ready for operation and our finger is aseptic.

In judging the symptoms, it should always be remembered that not all cardiac injuries present the same clinical picture, and that not all the classical signs appear in every case. The decision as to treatment and the framing of a diagnosis must therefore not be delayed until all the text-book symptoms supervene and the patient is moribund.

The *subjective sensations* experienced at the moment of the injury, often referred to as those of indescribable fear, are of significance. *Reflex signs* such as fainting and vomiting may follow immediately on the injury, but these must be distinguished from its mechanical effects, which will be referred to forthwith.

Apart from those rare cases in which the patient bleeds externally from his wound, there are two distinct forms of cardiac injury: (1) in which the pericardium and heart are alone involved, and (2) in which the pleural cavity is also opened.

In a *purely cardiac injury* the most striking features, in addition to a certain reflex pallor, are the cyanosis and dyspnœa of the patient. The pulse is weak, rapid, and markedly irregular; the heart sounds are feeble and appear to come from afar. The cardiac dulness is more or less increased, but the results of percussion and auscultation of the lungs are normal.

We have just said that the *cardiac dulness* is increased. This is true of most cases of purely cardiac injury, but the extent of the increase varies considerably. It must not be estimated by the standard which obtains in cases of pericardial effusion, as the inexperienced are apt to do. An acute hæmorrhage does not distend a healthy pericardium to the same degree as a gradually developing effusion. We can, therefore, only expect any considerable increase in the area of dulness after cardiac injuries in cases wherein blood is slowly oozing for days. In the very severe cases, on the other hand, death is too sudden to permit of the recognition of any striking distension of the pericardium.

Observation of the patient over a number of hours will probably show that moments of improvement alternate with periods of exacerbation which appear to come on in attacks, signifying that the heart temporarily recovers, only to succumb in the struggle against unfavourable mechanical conditions. The exacerbations get worse and worse, and the patient sinks unless hæmorrhage ceases spontaneously or is checked by suture of the heart.

This picture constitutes the condition known as *compression of the heart* by means of the blood enclosed within the pericardium. Whether the usual explanation of auricular compression is correct or not is an open question.

The following case will illustrate the foregoing remarks. A young melancholic stabbed himself three times in the cardiac region with a sharp file, and was brought into hospital three hours later with a miserable rapid pulse. During the examination dyspnœa and cyanosis became severe, the pulse inappreciable, and the eyes glassy. A rib was immediately resected, and the pericardium, which was full of blood, was opened. At this moment the nurse who was looking after the pulse exclaimed, "The pulse has come back again!" No hæmorrhage had occurred into the pleura. The signs were therefore not those of anæmia but of compression of the heart. The patient was taken back to bed with a good appearance and full pulse. The heart was not sutured because the hæmorrhage ceased spontaneously, and the patient recovered.

The picture is quite different when the **cardiac injury is complicated by injury to the pleura.** If the pleuro-pericardial wound is large enough the patient simply bleeds into the thoracic cavity and he presents the appearance of an acute anæmia. The patient looks pale rather than cyanotic, the cardiac dulness is very little enlarged, if at all, but an increasing liquid effusion takes place in the injured pleural cavity. The pulse is rapid, small and irregular, and the patient manifests the same momentary improvements and sudden exacerbations which occur in purely cardiac injuries. On auscultation the cardiac sounds prove to be feeble. Various valvular murmurs are audible, but nothing distinctive. A splashing murmur like the sound of the wheel of a water-mill is of more importance, because this is conclusive of the entry of air into the pericardium.

We must, however, not expect to find a *typical picture* in any individual case. If the pleuro-pericardial wound is small, blood may indeed escape from it into the thoracic cavity, so that the patient becomes to a certain extent anæmic, but the opening may be eventually closed by a clot, so that symptoms of heart compression will be present in addition to those of anæmia. In these intermediate cases it is not so important to diagnose all the details of the injury as to recognize the fact of cardiac injury as soon as possible, and appraise accurately the indications for surgical interference.

The following statement may be taken as a guide in practice:—

If an injury in a situation wherein the heart may be involved is followed by derangement in cardiac action or by acute anæmia, we must assume the probability of an injury to the heart, whether the cardiac dulness be increased or not. If the symptoms gradually increase despite transitory improvement, surgical measures must be adopted unless contra-indicated by external conditions.

"Unless contra-indicated by external conditions." The maintenance or strict asepsis is so important in these operations which usually concern the pleura and pericardium that cardiac suture ought only to be undertaken when all external conditions are favourable, unlike the rule for tracheotomy. If at all possible the patient should be conveyed to the nearest hospital. Experience shows that there is ample time for this if the diagnosis has been made without delay.

Do the foregoing remarks justify us in concluding that there is *no* cardiac injury in the absence of anæmia and circulatory disturbances? Certainly not. Even penetrating wounds may run their course without symptoms, and may remain unrecognized despite careful examination.

An old man stabbed himself three times with a kitchen knife in the cardiac area. Although the heart was most carefully examined no sign of injury could be detected. The patient died in eight days, from pneumonia, and the autopsy revealed a small incised wound which had gone right through the left ventricle, at the apex, but which had been sealed up by fibrin. The pericardium only contained a little blood-stained fluid.

Such a case is not one for immediate operation, but it requires careful watching, lest subsequent hæmorrhage necessitate an operation eventually.

A word of warning in conclusion. Too much zeal must not be evinced in this newly-acquired surgical province. Many uninjured hearts have been exposed, showing the necessity for most careful examination to avoid confusing an injury of some other thoracic organ with a cardiac injury.

CHAPTER XXIX.

THE SURGERY OF INFLAMMATORY DISEASES OF THE LUNG.

THERE are certain diseases of the lung in which the patient is entitled to the benefit of surgical treatment. Nothing demonstrates this so clearly as the action of a recently deceased physician who, doubting the efficacy of his prescriptions, betook himself to the knife and became one of the most experienced of lung surgeons.

The diagnosis of surgical diseases of the lung comes within the province of the physician and general practitioner, because these maladies are purely "medical" at first. The surgical sense of the practitioner should evince itself by recognizing the exact moment when resort to the knife is required.

Empyema, abscess of lung and gangrene of the lung are the diseases which are of main interest in this connection. Bronchiectasis and actino-mycosis of the lung only rarely lead to operative procedures. The surgical treatment of phthisis and of emphysema is in much too early a stage to justify any discussion here.

A.—EMPYEMA, ABSCESS OF THE LUNG, GANGRENE OF THE LUNG.

From an etiological standpoint the following possibilities exist :—

(1) If a **pneumonia** fails to resolve in the desired manner, or if pyrexia recurs after the crisis the first thought which enters one's mind is that of *empyema*. The diagnosis is made from dulness at the base, weakened respiration and loss of vocal fremitus and confirmed by exploratory puncture. A skiagram is almost superfluous. An empyema extending to the spine of the scapula, and unrecognized, must be ascribed to thoughtlessness on the part of the medical attendant. A careful observer will never be taken by surprise by an empyema bursting through into bronchi or thoracic wall. If an empyema is indicated by fever, dypsnœa and emaciation, but physical signs are absent, we need not doubt the accuracy of the diagnosis, but must search for the empyema in the correct situation. It may be *interlobular*, and therefore fail to yield the ordinary signs. A circumscribed area of diminished respiration and of dulness, generally with some adjacent bronchial breathing bounded about and below by normal lung resonance and well-preserved respiratory murmer, usually directs us to its position. A needle correctly aimed will demonstrate pus at once. (See under Sub-phrenic Abscess.)

But in such a case the differentiation from *abscess of the lung* is difficult. This is also a frequent sequel of pneumonia, and if not situated at the base has an area of more or less normal lung tissue below it. The greater extent of the dulness and the later occurrence or entire absence of perforation into the bronchi are, however, distinctive of interlobular empyema.

A skiagram may render some assistance in this differentiation, because a more or less transverse non-transparent area indicates an empyema, whereas a restricted roundish thickening points to abscess (fig. 136).

If the expectoration has a peculiar fœtid smell and contains shreds of lung tissue the case is no longer merely one of abscess; it has become gangrenous.

If *croupous pneumonia* has been the antecedent disease, as we have hitherto assumed, experience shows that some form of empyema is most probably in question.

(2) If the pneumonia has been caused by the inhalation of any

kind of **foreign body,** or of fluids, the conditions are different. It is quite true that empyema is not a rare sequel, but *abscess of the lung* is of more frequent occurrence, and *gangrene* often follows it.

As we have previously seen, foreign bodies include everything taken into the mouth foolishly by children and adults, and inhaled through carelessness. The consequent abscess may run a very chronic course and persist for years, even ten or more. I have seen a female, aged 40, suffering from a chronic pulmonary abscess and bronchiectasis which she rightly attributed to inhaling a bean in her youth.

It is also necessary to note the occurrence of the inhalation of fluids, especially in the vomiting of anæsthesia and in operations on the mouth and throat, for the removal of putrefying carcinomata, or for the extraction of teeth under general anæsthetics. The extraction of numerous teeth under anæsthesia is no trifling matter; nevertheless, the operation is, unfortunately, often undertaken when the patient is quite unfitted for it.

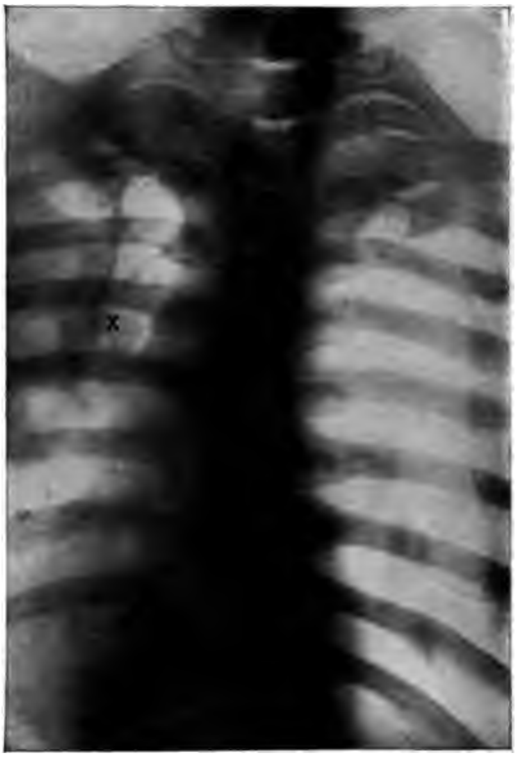


FIG. 136.—Skiagram of abscess of lung (X).

If some lung complication supervenes with these circumstances the diagnosis is generally easy. At first a pneumonia is diagnosed. If an effusion takes place in the pleural cavity puncture with a needle will show whether it is serous or purulent. If the sputum after settling separates out into three layers—the lowest of pure pus, then clear liquid and the highest of mucus—an abscess must be present. If we are met by a repulsive odour on entering the sick room, the case is either fœtid bronchitis or gangrene. The sputum in the latter case is greenish or brownish and putrid, separating into three layers, and contains greyish or dark shreds of necrotic lung tissue, or, at any rate, elastic fibres visible under the microscope. If repeated examinations fail to reveal any of these structures, we must content ourselves with the diagnosis of fœtid bronchitis.

(3) In a third group of cases the lung symptoms are preceded neither by the inhalation of a foreign body nor by pneumonia. The disease follows some inflammatory process which may possibly be situated at some distance from the lung; and the exciting cause of the infection reaches the lung by way of an **embolism.** Any inflammatory process may serve as the cause, *e.g.*—sore throat, a furuncle, suppuration within the abdominal cavity, especially in the region of the female genitalia, or an infective thrombo-phlebitis. If a few organisms only, or very minute particles of infected material are conveyed, the result will be, according to their localization, either a pleurisy or multiple small abscess formation with signs of pneumonia. If, on the other hand, a larger infective thrombus reaches the pulmonary artery a more or less extensive focus of gangrene will result.

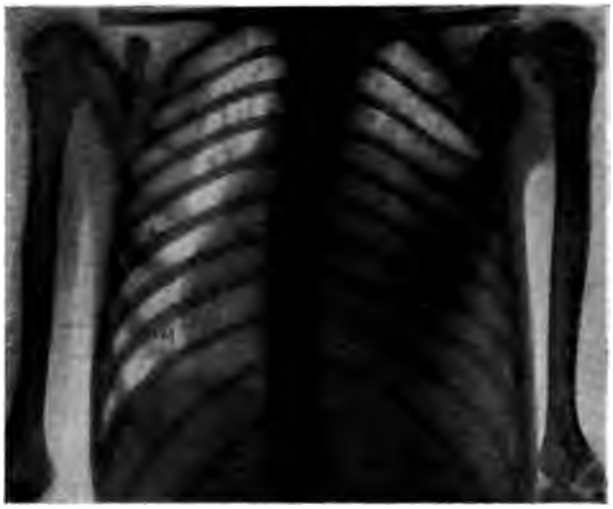


FIG. 137.—Meta-pneumonic empyema on right side (posterior view).'

The history and physical examination, as have been seen, usually suffice for a correct diagnosis. If an empyema is confirmed by puncture the indications are for an immediate evacuation by surgical measures either by simple syphon drainage or by the more reliable method of rib resection. If we suspect an abscess of the lung or a gangrenous area, we must be more careful in regard to puncturing, because the pleural cavity is probably still free. We must therefore rather resort to a skiagram or a screen examination, and only proceed to puncture when everything is ready for an operation. It is even better to abandon the puncture altogether, or, at any rate, until the lung is exposed. The nature of the expectoration also testifies to suppuration, and its position is more correctly estimated by means of physical examination and a skiagram than by puncture.

We have hitherto assumed that the diagnosis of disease within the pleural cavity can be made with absolute certainty; but this is not always the case. In discussing subphrenic abscess we shall see that it is very easy to mistake this for an empyema, especially on the right side. The diaphragm may be so tightly pressed against the thoracic wall, and the lung pushed up so far that although the needle shows the pus to be situated apparently in the sixth or seventh intercostal space, the abscess is really below the diaphragm. On the other hand, a circumscribed diaphragmatic pleurisy in the depths of the inferior surface of the lung may be mistaken for a subphrenic abscess.

B.—BRONCHIECTASIS.

Bronchiectasis is either of congenital origin or the result of some circumscribed inflammatory process of the lung or pleura, such as pneumonia or abscess.

The diagnosis is based upon the paroxysmal attacks of expectoration, amounting sometimes to a whole mouthful, especially in the morning; to the separation of the sputum into three layers—after standing, and to the presence of the particles of pus known as Dittrich's plugs. On physical examination moist râles are heard in cases of small bronchiectasis, and when large circumscribed cavities are present the signs are those of infiltration and cavitation. A skiagram should always be taken, because we may derive therefrom important conclusions regarding the situation and extent of cavities, if not about the very nature of the disease (fig. 143). The favourite situation of bronchiectasis is in the left lower lobe.

In the diffuse form there is only evidence of thickening of the lung tissue, which is indistinguishable from a severe tubercular infiltration; but in saccular bronchiectasis a cavity which is hardly distinguishable from a tubercular cavity may be visible.

It is well know that clubbed fingers develop after some years in cases of bronchiectasis, but this condition may also be due to quite different causes, and is, therefore, not of much value from a diagnostic standpoint.

In the differential diagnosis of bronchiectasis care must be taken to distinguish it from *pulmonary abscess* and from *tubercular cavities.* If there are no tubercle bacilli in the sputum the latter may probably be excluded. Pulmonary abscess differs from bronchiectasis in that it supervenes directly after the primary disease, *e.g.*, pneumonia or inflammation caused by a foreign body, whereas bronchiectasis takes months or years to develop. If there has been no causative disease, congenital bronchiectasis is the most probable diagnosis. A differential diagnosis is important, in order to determine the surgical procedure to be adopted.

C.—ACTINOMYCOSIS.

The early stages of actinomycosis of the lung are either entirely overlooked or confused with tubercle. If the patient has a chronic cough with purulent sputum and elevations of temperature, he will generally be diagnosed as a case of phthisis—and justly so. But there are certain signs which ought to suggest the possibility of actinomycosis even at this stage. These consist of a localization of the disease to the middle and lower lobes, leaving the apices free, and also the incidence of pleuritic attacks with simultaneous drawing-in of the chest. A positive diagnosis may be obtained by examining the expectoration, at any rate if it contains the actinomycosis granules. The diagnosis is easier when the disease reaches the thoracic wall and becomes evident on the surface. We shall return to this in a subsequent section, but would only add here that operative treatment of this disease has affected a complete cure in many cases.

CHAPTER XXX.

TUMOURS AND ALLIED GROWTHS WITHIN THE THORAX.

PATHOLOGICAL growths within the thorax naturally fall under two groups: (1) Those of the mediastinum; (2) those of the lungs. Each group possesses its characteristic symptom-complex, and will therefore be discussed separately.

There are transitional forms here, as elsewhere, which introduce difficulties in diagnosis. Thus a small cancer of the lung with enlarged mediastinal glands will yield the symptoms of a mediastinal tumour, whereas a sacculated aneurism growing, towards one lung will suggest the possibility of a pulmonary tumour.

(1) MEDIASTINAL TUMOURS.

No region is so inaccessible to direct examination as the mediastinum, and nowhere do tumours sail so long under false colours as here. The diagnosis is only made under a feeling of great responsibility, because the impotence of therapeutics renders the diagnosis a sentence of death.

Most of the new growths of the mediastinum chiefly involve the lung or the larger bronchi. An *irritating cough*, without expectoration, and *dyspnœa* are, therefore, the symptoms which dominate the clinical picture for a considerable time and suggest some form of pulmonary disease—phthisis in young people, chronic bronchitis and emphysema in elderly patients. If there is, in addition, *paralysis of the recurrent laryngeal nerve*, the condition is looked upon with more gravity, although this form of paralysis may exist without any serious significance. If careful attention is bestowed on the form of the dyspnœa, it will often be noted that an attack comes on in certain postures of the body and that it ceases in other postures. The consideration of this symptom should suggest the possibility of some mediastinal growth, provided there is nothing in the neck like a goitre or other tumour to account for it. If the *veins of the neck gradually dilate*, and a collateral venous anastomosis develop on the chest wall, while the face looks puffed and the neck distended, our suspicion will

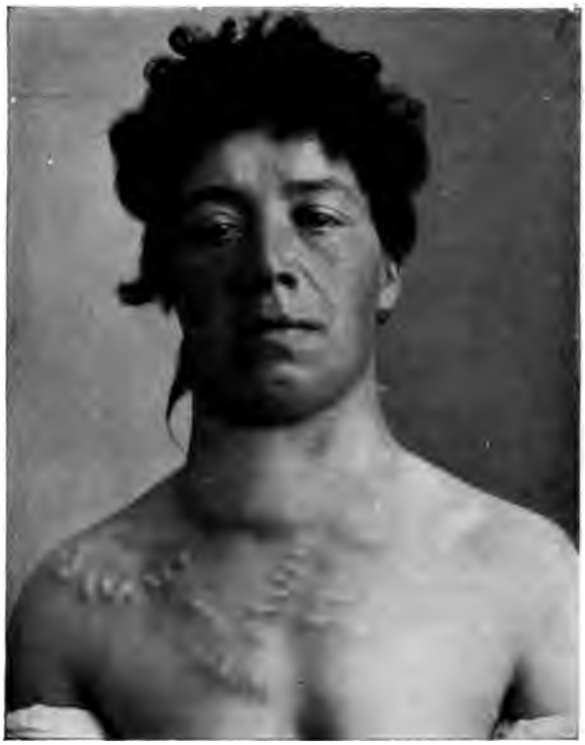


FIG. 138.—Diffuse colloid goitre with a considerable part inside the thorax (see fig. 139). Pronounced caput medusæ. Dyspnœa.

secure new support. At this stage there will generally be somewhere in the thorax, especially over the sternum, an abnormal *dulness on percussion*, and the aid of the *X-rays* should also prove of service. As the tissue of the tumour is less transparent than lung tissue, a mediastinal growth of any appreciable size, of whatever nature it be, will throw a superimposed shadow on one side or other of the sternum. The peculiarities of this shadow often afford further diagnostic assistance and provide definite conclusions as to the nature of the growth. But other clinical methods will furnish information on this head.

Let us consider the main possibilities: In infancy the most likely condition is *hypertrophy of the thymus gland*, or some unusually great *hyperplasia of the bronchial glands*. In older patients we endeavour to decide beween an *aortic aneurism* or a genuine *tumour*. Innocent tumours mainly consist of intra-thoracic goitres, dermoid and hydatid cysts, and malignant tumours comprise carcinoma and sarcoma, which may arise from the thymus, bronchi, lymphatic glands or connective tissue. Cancer of the œsophagus, which should properly be included here, is generally fatal before reaching dimensions which bring it in the catagory of mediastinal tumours.

The early recognition of **hypertrophy of the thymus** is particularly important, because many cases have been successfully operated on.

Hypertrophy of the bronchial glands in scrofulous, or rather in tubercular children, is only important surgically as a matter of differential diagnosis. Without a skiagram the diagnosis is usually nothing more than a suspicion, unless opportunely there are such definite signs as glands in the neck, dulness over the sternum, and paralysis of the recurrent laryngeal nerve. The skiagram, however, shows these glands with the clearest distinction, and should, therefore, always be procured whenever this diagnosis is entertained. If, however, the hypertrophy be slight, the significance of the skiagram may remain doubtful. It is most important to avoid the error of mistaking the normal structures—vessels and bronchi—seen at the root of the lung for mediastinal glands. The diagnosis is aided by the invariable presence of some calcification which reveals itself by casting a dark shadow.

In adults the differential diagnosis mainly lies between **tumour and aneurism.** Certain conclusions may be drawn from the shape of the area of dulness. In large aneurisms the dulness projects in front, to one or both sides of the upper part of the sternum, and it appears posteriorly chiefly in the region of the left and sometimes of the right upper lobe of the lung. Mediastinal tumours may present the same condition in regard to dulness, but they do not adhere to any rule.

If the thoracic wall bulges and pulsates, and the hand feels a definite thrill, and if a souffle is heard with the stethoscope, the diagnosis requires no great skill, especially if the radial pulses are unequal and there is a history of syphilis (fig. 142). But sometimes nothing is heard on auscultation, the radial pulse is equal on both sides, and there is no previous history of syphilis. The actual symptoms, dulness, dyspnœa, paralysis of the recurrent laryngeal, intercostal pain, and possibly also inequality of the pupils due to pressure on the oculo-pupillary fibres of the sympathetic (to be examined in semi-darkness), the emaciation and even the shape of the demonstrable dulness might just as well be due to a mediastinal tumour, or, in exceptional cases, to greatly enlarged tubercular bronchial glands. We must, therefore, be guided by indirect diagnostic considerations. The symptoms of an aneurism usually come on very slowly, taking years to develop, and the patient is quite unconscious of them. Unless a sudden rupture occurs their progress is very gradual. A mediastinal tumour, on the other hand, develops more rapidly, and after having once given rise to symptoms proceeds uninterruptedly to the end.

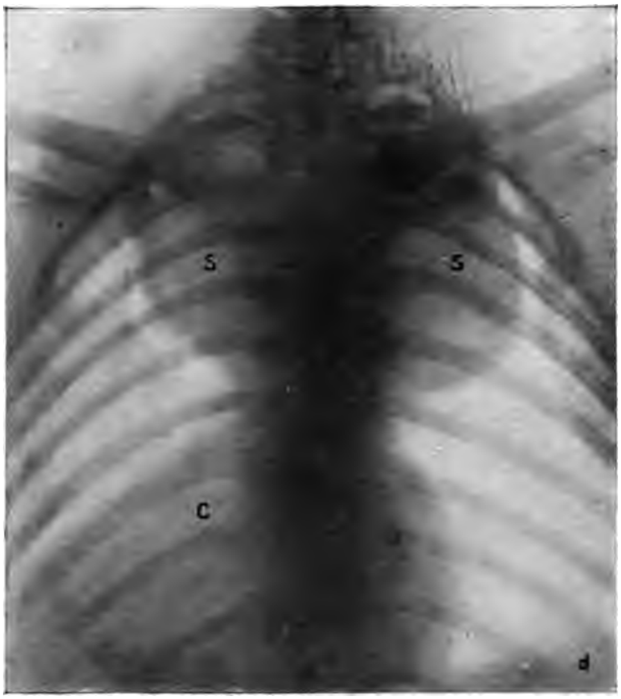


FIG. 139.—Intra-thoracic goitre. *S—S*, the goitre; consisting of both lower cornua, as shown by the operation. *c* = heart, *d* = diaphragm and liver.

Sometimes we are led upon the right track by the condition of other organs. Thus we may conclude from the presence of enlarged glands in the neck, axillæ or groins, that the mediastinal disease also depends upon enlarged glands. The history of the case, the other clinical symptoms, and more especially the examination of the blood, will indicate whether the glands are leukæmic, pseudo-leukæmic, or tubercular in character. Sometimes it may be impossible to determine this point until a gland has been excised from the neck and examined microscopically.

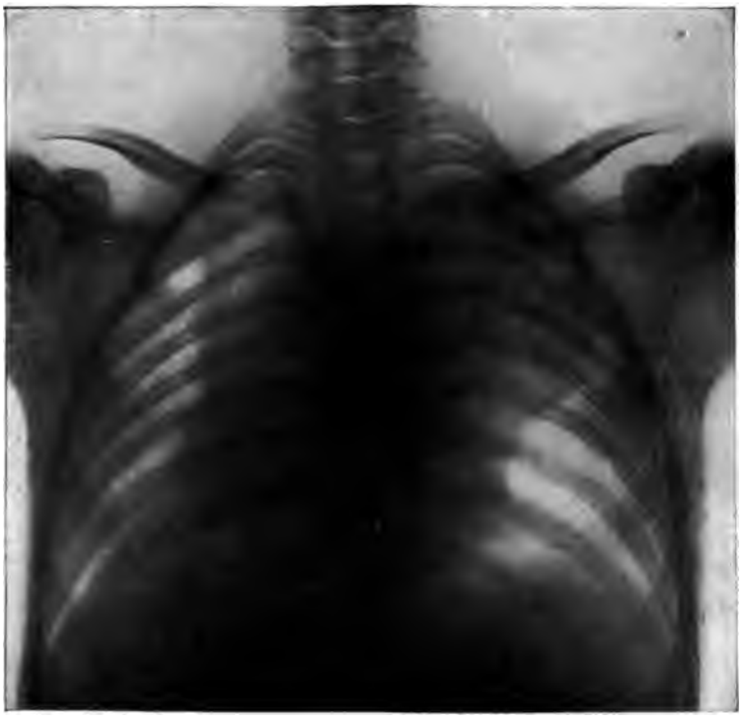


FIG. 140.—A mass of mediastinal glands encroaching on the right lung in a case of lymphadenoma

It may be pointed out that Cohnheim's variety of pseudo-leukæmia is most probably present, if there be an extensive development of the mediastinal glands, with histological features consisting of a simple increase of lymphadenoid tissue, and if, at the same time, the blood condition is approximately normal. Leukæmia is diagnosed from the blood condition, and Sternberg's variety of pseudo-leukæmia (lymphogranulomatosis) is recognized by its special histological features.

In the absence of any such indication, we should think of a primary growth in the mediastinum, or a metastasis of some other unrecognized primary growth (*e.g.*, a hypernephroma).

Skiagraphic examination is often absolutely decisive.

In the case of an aneurism a thick, sharply-limited, rounded shadow is seen. If the aneurism arises from the *ascending aorta* or *aortic arch*, the shadow is situated above the heart, fitting over it like a cap, corresponding to the dulness previously described (fig. 142). If it is an aneurism of the *innominate artery* the shadow is situated to the right of and above the aortic arch, and is not distinguishable from a saccular aneurism arising from the arch and growing towards the right. A semi-circular shadow on the left side, situated much lower down, indicates an aneurism of the descending aorta, if the shadow can be distinctly defined separately from the heart shadow. The penetration of an aneurism through the thoracic wall can be recognized clearly in the profile. All this is visible in the skiagram, but the screen allows us to see the pulsatile movement of the margins of the shadow. The absence of this pulsation is, however, no proof *against* aneurism, because there are such things as non-pulsating aneurisms; but the presence of an extensive pulsatile dilatation of the margins of the shadow, especially if bilateral, may be regarded as positive proof of aneurism. But if the pulsation is unilateral only or if it is limited to one place the case may be one of new growth pushing up the aortic arch.

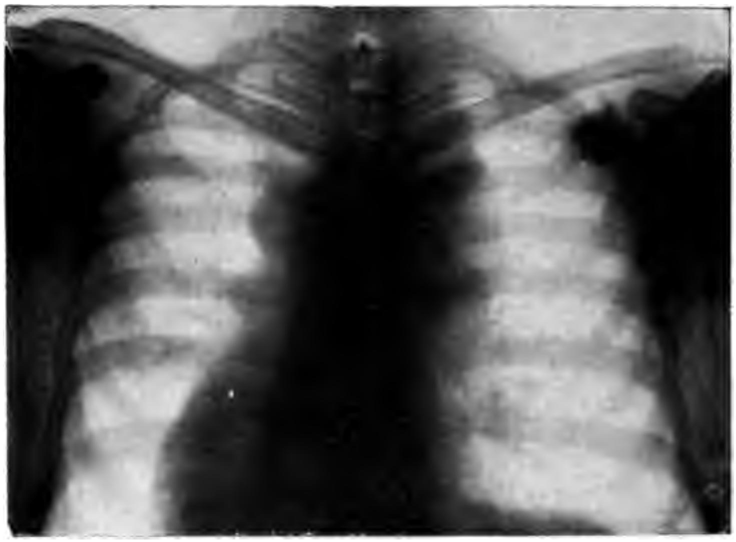


FIG. 141.—Mediastinal tumour. Metastasis from a hypernephroma which produced no clinical symptoms.

The shadows cast by mediastinal and pulmonary tumours are quite different to the typical shadow of aneurism. Like the dulness produced by these tumours, their shadows have no typical situation, but they invade irregularly the median shadows cast by the spinal column and sternum, either to the right or left, encroaching into the lung area. Their limits are less sharp than in cases of aneurism (fig. 141), the border line especially being less regularly defined. Sometimes it is possible to see quite clearly that the growth is composed of separate nodules. We shall see later that there are exceptions to this rule.

The following case illustrates the considerations which lead up to the diagnosis of aneurism.

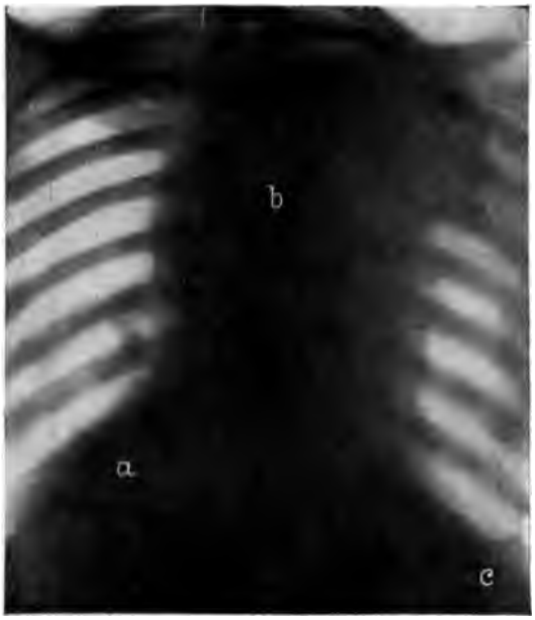


FIG. 142. Aneurism of the aortic arch. *a* heart, *b* aneurism, *c* diaphragm and liver (seen from behind).

An alcoholic male, aged 42, who had been losing flesh for some time, suffered from indefinite general malaise, which was attributed to a cirrhosis of the liver, with which he was afflicted. During the last few weeks striking dyspnœa and hoarseness developed, and the patient came into the consulting room emaciated and short of breath. He might have been taken for a case of advanced phthisis, had he not stated that he had had no cough up to quite recently, and even now there was no expectoration suspicious of tubercle. The breathing was rapid, but auscultation disposed of the idea of pulmonary disease. Cardiac action was rapid, but the heart sounds were pure. The dyspnœa could not be accounted for by pulmonary disease or by valvular defect. The clinical picture did not fit in with disease of the heart muscle. The only explanation was offered by a slight dulness on both sides of the upper half of the sternum, which might be caused by an aneurism or by a tumour. The radial pulse was equal on both sides. Röntgen-ray examination revealed a shadow, corresponding in form to an aneurismal shadow, above the heart, reaching to the throat. There was, however, no distinct pulsation, and it was proposed that the patient should come into the hospital for further examination of this point and for closer observation. But, instead of coming to the hospital, his doctor reported, in a few days' time, that the patient had died from hæmorrhage within a few minutes.

If an aneurism manifests itself for a considerable time, merely by a single symptom which is not very typical, much difficulty may be encountered before a diagnosis is established. Thus, I saw a patient who had been treated for two years for intercostal neuralgia, the explanation of which was not revealed by physical examination of the thoracic organs. Resection of three intercostal nerves afforded temporary relief. The recurrence of the pain suggested an X-ray examination, and then an aneurism was discovered !

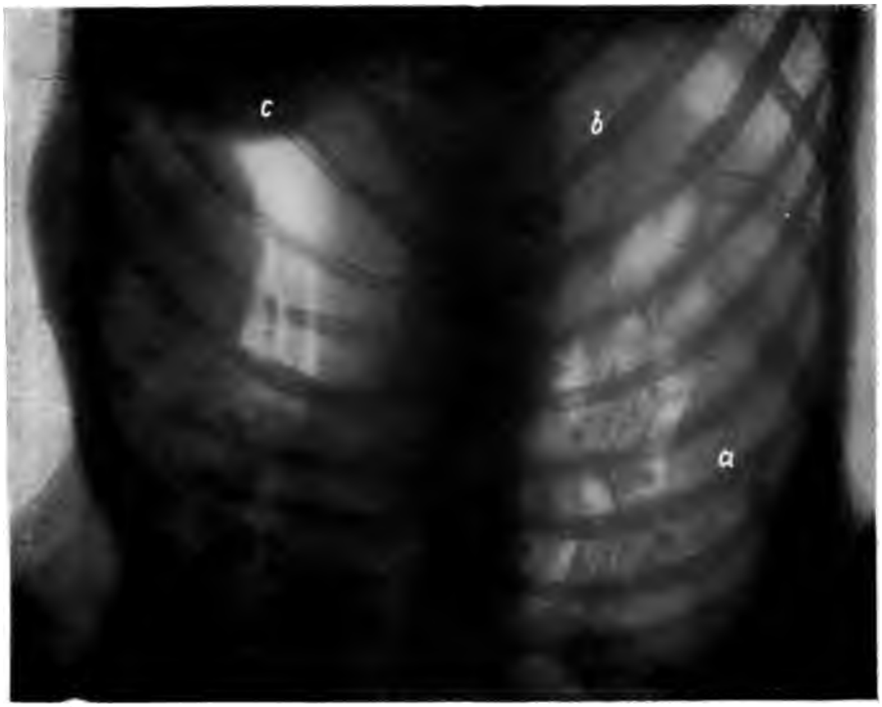


FIG. 143.—*a* = Cancer of the right lower lobe penetrating through the thoracic wall. *b* = Diffuse bronchiectasis of the right upper lobe. *c* = Old tubercle of the left upper lobe.

Such cases show how easy it is to overlook an aneurism. We do not, however, mean to suggest to certain journalists that they should diagnose "ruptured aneurism" any more frequently than they do at present in cases of sudden death.

If, after considering all the diagnostic signs of aneurism, we exclude this condition and diagnose a **new growth,** our next step is to determine its nature.

We have already mentioned intra-thoracic goitre, dermoid and hydatid cysts as innocent tumours. A **goitre** is easily recognized if it is merely a continuation of one, visible and palpable in the neck (deep goitre). The diagnosis is, however, more difficult if the whole goitre is concealed within the thorax, and the corresponding lobe of the thyroid gland cannot be felt or is only rudimentary (pure intra-thoracic goitre). In such cases the goitre originally develops from an inferior cornu of the thyroid gland, extending very low down. The goitre grows into the thorax, and becomes so large that it can no longer slip out, and as it grows it drags the rest of the thyroid lobe down with it. It may also originate, as a true or false secondary goitre, from an accessory thyroid gland.

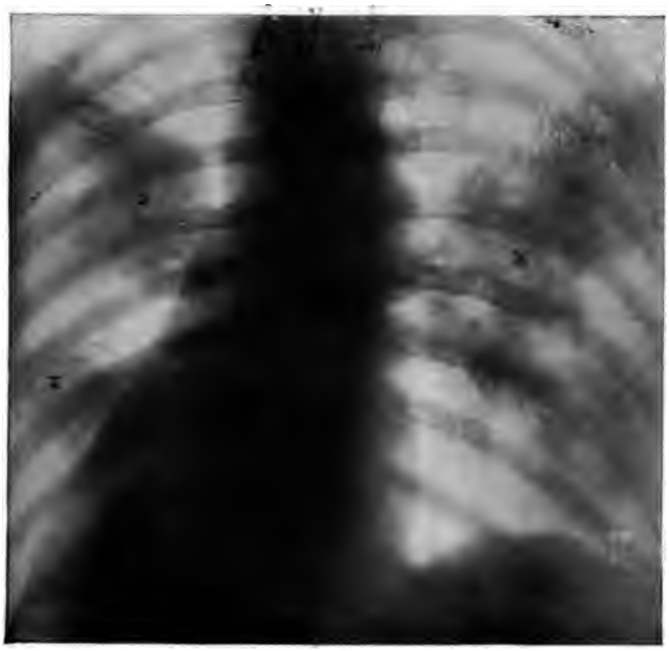


FIG. 144.—Metastatic cancer of lung. x x = foci in both lungs.

A female, aged 68, who had been suffering from bronchitis for many years, came to the hospital because of extreme dyspnœa. She could only breathe in a sitting and bent-up position. There was dulness on both sides of the sternum, and a skiagram showed a sharply defined shadow, like a cap, above the heart, reaching as far as the throat, which suggested an aneurism by its shape. But its edge did not pulsate, and other evidences of aneurism were also wanting. The right lobe of the thyroid gland contained a few small colloid nodules. Nothing could be felt of the left lobe of the thyroid, except some indefinite resistance in the throat. The case, therefore, appeared to be one of intra-thoracic goitre, which would also account for the bronchitis. The operation confirmed this diagnosis, and the removal of the goitre permanently cured the malady. The operation in the case depicted in fig. 138 was followed by the same result.

Dermoids of the mediastinum are mostly situated behind the manubrium, and are accessible to operative treatment. The diagnosis has occasionally been based on the expectoration of hairs, after perforation of the dermoid into a bronchus. Otherwise we cannot get much beyond suspicion.

Hydatid cysts will only be thought of in districts where they are endemic, and then the diagnosis is something of a guess, unless the nature of the disease is betrayed by attacks of urticaria.

Malignant tumours are recognized by their rapid growth and the correspondingly swift increase of all symptoms. They are also indicated by the significant extent and rapid increase of the dulness. We have already referred to their skiagraphic appearances. They are usually **sarcomata,** originating either in lymphatic glands or connective tissue.

(2) TUMOURS OF THE LUNG.

Tumours of the lung, like those of the mediastinum, consist of **dermoids, hydatids, carcinomata, sarcomata,** and of **chondromata,** the last starting in the bronchial cartilage. In their early stages all these growths, if they give rise to symptoms at all, are mistaken for tuberculosis. A dermoid is only recognized when hairs are coughed up, a hydatid only when it bursts into a bronchus and causes suffocation or when this occurs under the very eyes of the unsuspecting practitioner as a result of an exploratory puncture. If the patient does not succumb the microscopic examination of the fluid, and the occurrence of urticaria consequent upon the puncture will establish the diagnosis. The skiagram, with its sharply defined spherical shadow, is very significant of hydatid cyst. If the symptoms do not accurately fit in with those of tuberculosis we should think of a malignant growth, especially if the patient is expectorating reddish fruit-juice sputum. Sometimes cancer can be recognized by particles of tissue in the expectoration. Malignant disease of the lung or bronchi is more likely to be carcinoma than sarcoma, but even carcinoma is quite rare.

According to Schwalbe, the presence of stridor is in favour of sarcoma, and its absence suggests carcinoma, because in sarcoma the bronchial glands undergo more enlargement than in carcinoma.

The accompanying illustrations of secondary cancerous nodules in the lungs show how clearly tumour areas can be marked out by means of X-rays.

It is of interest to note the occurrence of lympho-sarcoma of the lungs in miners, who inhale arsenic-containing dust.

CHAPTER XXXI.

SWELLINGS AND TUMOURS OF THE THORAX.

SWELLINGS and tumours on the surface of the chest originate from one of the thoracic viscera, usually the lung or pleura, or from the chest wall itself. It is important to arrive at some decision on this fundamental point before making a physical examination, and this can be done by obtaining a careful clinical history.

A.—PRIMARY DISEASE WITHIN THE THORAX.

If the appearance of a tumour on the chest wall is the final episode of a long history of illness, which began with an irritating cough without expectoration, dyspnœa and hoarseness, and which was followed by disorders of the circulation, we must think of a lung or mediastinal tumour, an aneurism or of some inflammatory condition.

We must think of the possibility of aneurism because an exploratory puncture, thoughtlessly undertaken, may place us in a very awkward predicament.

We have already dealt with the important points which concern tumours of the mediastinum and of the lung.

A swelling appearing in the thorax after a disease which began with *inflammatory symptoms*—either of an *acute* character like pneumonia, or of a *chronic* character like a slow pleurisy—is suspicious of a pleural empyema which has made its way to the surface, a so-called **"empyema of necessity."** Nowadays we lay more stress on the "necessity" of operative treatment before the pus has become subcutaneous.

We will give three typical cases illustrating the most important forms :—

(1) The empyema which has burst through may be of an **acute infective** variety.

A middle-aged man suffered from a circumscribed pneumonia, with which signs of pleurisy were associated. A diffuse, phlegmonous, rapidly extending swelling at the back indicated the urgency of surgical aid. Examination showed that there was an effusion into the left pleural cavity with a phlegmonous swelling of the soft parts on the corresponding thoracic wall. Diagnosis : ruptured empyema. This was confirmed by operation. The rupture was due to a cause which is in no way unique. Two days beforehand the pleura was punctured for bacteriological diagnosis, and evidently this afforded an opportunity for the introduction of infection from the soft parts along the puncture wound. This is a lesson that in acute cases operation should immediately follow puncture, should pus be discovered.

(2) Another variety is of **tubercular** origin.

A young man, whose previous history pointed to tubercle, complained about the gradual development of a painless swelling, about the size of a goose's egg, immediately to the right of the sternum. The swelling could not be displaced, and it did not undergo any change in volume with respiration, indicating some connection with the interior of the chest. But there were definite evidences of tubercular disease over the right upper lobe of the lung, which led to the conclusion that there was some direct connection between the two conditions. Operation showed that the superficial abscess was in direct connection with an encapsuled collection of pus within the thorax.

If a cavity ruptures externally the swelling will evidently contain gas. There is always some secondary infection in a cavity, and this confers acute inflammatory characters upon the abscess, as happens when a purely tubercular collection of pus breaks through.

We shall subsequently enter into the details of differential diagnosis between a tubercular empyema which has burst through, and tuberculosis of the ribs.

(3) The third possibility is illustrated by the following case :—

A young girl, suffering from symptoms of chronic pleurisy, was admitted to the hospital with a bilateral effusion. The needle met with pus on the left side, but not on the right. Resection of the ribs, on the left side, was performed. The chronic course of the disease and the curious appearance of the pus suggested tubercle at first; but the pus really contained filaments similar to actinomyces, without granules. After a little while a swelling appeared in the anterior axillary line parallel with the ribs. At the periphery it was board-like in consistence, without any reddening of the skin, whereas in the middle it was soft and red. Even without the bacteriological examination of the left side these appearances would have strongly suggested actinomycosis. As a matter of fact, the pus from the right side contained the characteristic granules in large amount. Complete recovery ensued after treatment with potassium iodide.

Here, as in all cases of **pulmonary actinomycosis,** the first thought was of tubercle. This rare disease can only be identified by the discovery of the actinomycotic filaments or the characteristic granules in the sputum or pus, unless the board-like infiltration previously described, which indicates that the process has reached the surface, betrays the correct diagnosis to the experienced eye.

B.—PRIMARY DISEASE OF THE THORACIC WALL.

If nothing in the history or physical examination points to disease of the thoracic viscera, we must assume that the structure under investigation arises from the *bony wall of the chest* or from its *integuments.* We are not here concerned, however, with tumours of the mammary glands, as these are dealt with in a separate chapter.

(1) ACUTE DISEASES.

Acute swellings need not detain us long, as they rarely occur on the chest. The principal one is **acute osteomyelitis** of the scapula or clavicle, which can hardly be mistaken for anything else. The sudden onset with rigors and high temperature indicate the nature of the disease, and the bone affected is shown by the position of the swelling and the pain on pressure. An acute osteomyelitis of a rib, which is very rare, might be mistaken for an empyema which has broken through, but the distinctive antecedent symptoms of the latter would not have been present.

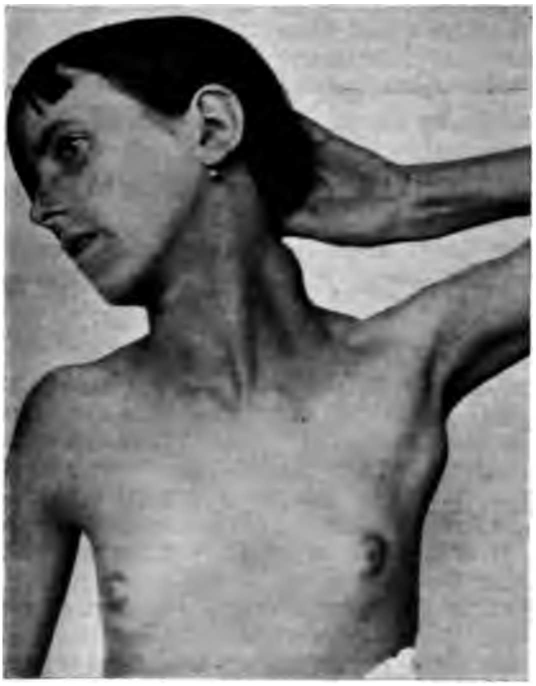


FIG. 145.—Tubercular glands of neck and axilla.

Phlegmonous processes are not of rare occurrence in the vicinity of the axilla. They generally originate in *lymphatic glands*, the infection being introduced from the periphery. The experienced observer will at once examine the *fingers*, and look on each one for some lesion, however insignificant.

Sometimes red streaks of lymphangitis lead towards the original wound. Frequently the wound is already healed by the time an abscess has developed in the axilla.

Occasionally very deep axillary abscesses form, as a result of *acute pustules* and *furuncles* which are not infrequent in this region. Purulent inflammation of the *sweat glands* (hydro-adenitis) should also be mentioned.

(2) CHRONIC DISEASES.

In a *gradually developing swelling* our first endeavour is to decide whether it is inflammatory or a new growth. As cystic tumours, apart from those of the breast, occur very rarely on the thorax, fluid contents point to pus. The only difficulty is to be sure of the fluid because it is not always easy to differentiate the fluctuation of fluid in small tumours from the soft elastic consistence of a lipoma, for example. The beginner is apt to confuse fluctuation with this soft elasticity, even in the case of larger tumours. When in doubt between a lipoma and an abscess, one should remember that a lipoma is characterized by a lobulated structure and numerous slight puckerings of the skin, whereas the skin over an abscess is quite smooth. The doubt can at once be solved by a puncture, but this should be left to the end of the examination if it is indispensable. If suppuration has not yet occurred an inflammatory origin would be indicated by spontaneous pain and tenderness on pressure.

(a) Chronic Inflammatory Processes.

Tuberculosis and *syphilis* are, with few exceptions, the principal causes of chronic inflammatory conditions on the thorax, whether the swelling be non-suppurative or whether it be an abscess. The inflammation may start in one of three tissues, viz., *lymphatic glands*, *muscles*, or *bones*.

Chronic inflammatory processes starting in the *lymphatic glands* are of a tubercular nature. They are situatéd in the neighbourhood of the axilla—sometimes in front, in the infra-clavicular fossa, sometimes below, between the anterior and posterior axillary line, and sometimes behind, under the scapula. The infra-clavicular glands cannot as a rule be felt separately, like the cervical glands, because they are situated under a thick layer of muscle. When they are diseased they present the appearance of a diffuse swelling of the deeper tissues, firm at first, but eventually declaring itself as an abscess which reaches the surface. But tubercular glands in the axilla feel exactly like those in the neck, and are therefore easily recognizable. The axillary glands are, however, rarely affected alone. The cervical glands are, as a rule, also involved (fig. 145). As in the case of the neck, there is also here the liability of confusing tubercle with malignant lymphoma; but we have already referred to the differential diagnosis in discussing tumours of the neck.

If an inflammatory area is situated within a **muscle**, the condition is usually *tubercular*, and rarely *gummatous*. It manifests itself as a painful hard little tumour, and its intra-muscular situation is easily recognized by the fact that it is freely movable when the muscle is relaxed, and is quite fixed when the muscle is contracted.

But most of these inflammatory processes arise from the **bones**, and all the bones of the thorax and shoulder girdle participate therein; the clavicle is, however, rarely at fault. There is one important distinction in regard to disease of these various bones, for, whereas disease of the superficial bones, like the ribs, sternum, and parts of the shoulder blade, may be recognized in the earliest stage before an abscess develops, spinal caries is only diagnosed after an abscess has formed, unless attention has been directed to the matter by functional disturbances. In these cases the abscess has often tracked a considerable way before reaching the surface.

A swelling of the *clavicle* of gradual origin should at once suggest **malignant new growth,** because tubercle and gumma are of very rare occurrence there. If no other new growth is discoverable we must regard it as a primary **sarcoma.** But microscopic examination will often surprise us, and indicate that a primary **carcinoma** is concealed somewhere. Cancer of the thyroid, breast or prostate should suggest themselves, because their secondary deposits preferably affect the bones; but tumours of other glandular organs, such as the gastro-intestinal tract, should also be thought of.

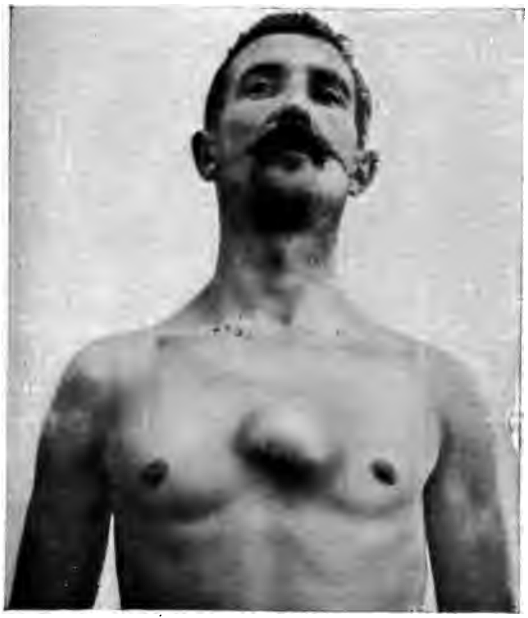


FIG. 146.—Tubercle of the sternum.

If there gradually form over a *rib* a spindle-shaped swelling, rather painless in itself, but still sensitive on pressure, we should at once think of **tubercle,** but even here an error is possible. Sometimes a **gumma** of the periosteum of the rib may have features very similar to tubercle.

Primary disease of the marrow (as shown by skiagram) points to tubercle; primary periostitis is not decisive.

The following case is important :—

A young man was admitted to the hospital with a spindle-shaped, somewhat sensitive swelling at the junction of the fourth rib and cartilage. The case had been diagnosed as tubercle, and the patient had been treated with iodoform injections. An affirmative answer was given to the question whether he had suffered from *typhoid fever,* and he added that the swelling started a few weeks after his recovery therefrom.

It is quite conceivable that typhoid fever may excite tubercular disease when a predisposition exists towards it—I have seen this in lymphoma of the neck. But such an assumption was out of the question in this case. We know that **post-typhoid osteitis and chondritis of the ribs** represent a special type of disease, with a very chronic course. Sometimes recovery occurs spontaneously, sometimes only after the extrusion or operative removal of a bony or cartilaginous sequestrum.

On the *sternum* the diagnosis lies between **tubercle** (fig. 146) and **gumma,** but we should also think of **malignant new growth** as long as no suppurative softening of the tumour has occurred.. The differential diagnosis is, however, difficult, and Kuster has removed a large gumma in mistake for a sarcoma. I have repeatedly seen tubercle give rise to swellings and enlarged mediastinal glands, which were very suggestive of sarcoma.

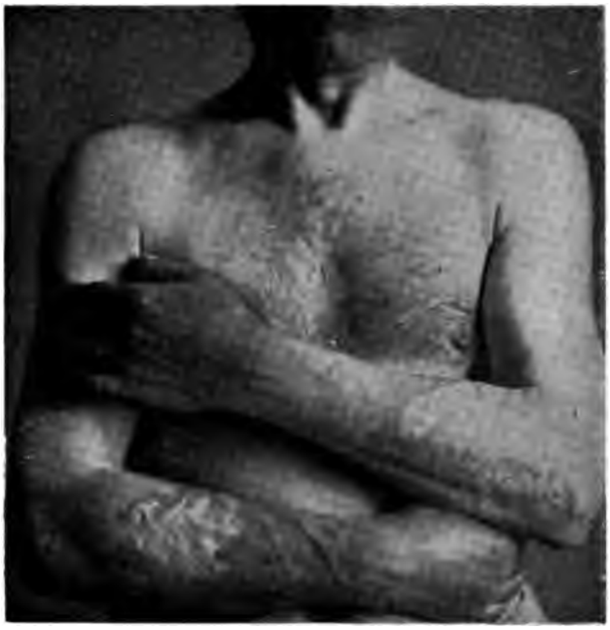


FIG. 147.—Gumma of the sternum and tertiary syphilitic scars on both forearms.

The statement that flaky, very liquid pus points to tubercle, and that viscous mucoid pus points to gumma is not borne out in practice.

Swellings of gradual origin on the *shoulder blade* are usually **tubercular** or **sarcomatous.** In the early stages there may be some serious difficulty in diagnosis, because adjoining the hard places

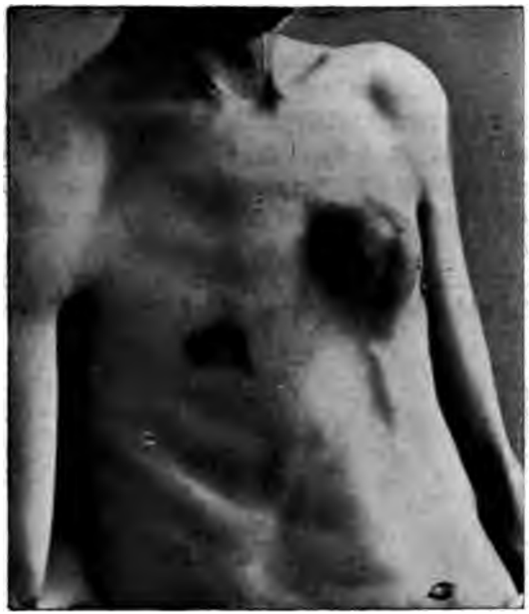


FIG. 148.—Thoracic wall, perforated by aneurism of ascending aorta.

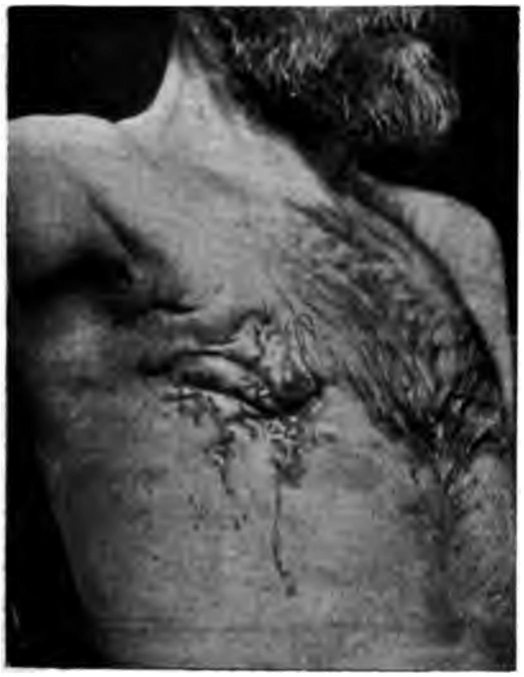


FIG. 149.—Tertiary syphilis of ribs.

are to be found soft portions, which resemble abscesses, a circumstance which also occurs with tumours. But when suppuration takes place in tubercle it becomes evident, in a short time, and often becomes quite extensive; so that in every case where the diagnosis is doubtful we must suspect sarcoma and treat accordingly. Anyhow, an early exploratory incision allows radical measures to be undertaken in a case of tubercle, and offers the only prospect of cure in sarcoma.

A history of *injury* is of no special value either for the one or the other diagnosis, because an injury may excite either tubercle or sarcoma.

If a cold abscess on the back appears to have no relation either with a rib or the shoulder blade, it probably arises from a *vertebra*—from the transverse process, vertebral arch or spinous process.

It is easy to decide whether the abscess is above or beneath the fascia by making the muscles of the back contract, but this is no great aid to diagnosis, because an abscess originating in the bone often reaches the surface.

The classical signs of spinal caries (which see) are of much more importance for its diagnosis than the variable behaviour of the abscesses to which it may give rise. These signs are muscular fixation of the spine (rigidity), pain on axial pressure—not always—curvature, and local sensitiveness to pressure. The last symptom is the one chiefly present in tubercle of the posterior portion of the vertebra, which is concerning us just now, the pain on pressure being manifest on the affected spinal process. But this sign is only of value if the sensitive spine is not contiguous with the wall of the abscess, but lies above it. In the absence of any sign of spinal caries we cannot decide whether the disease arises from the vertebra or from the posterior segment of a rib, unless a skiagram clears the matter up. But if this also leaves us in the lurch we must conclude in favour of the more frequent occurrence, *i.e.*, spinal caries.

(b) Tumours.

We now proceed to those morbid structures which are recognized as **tumours,** without any hesitation.

The innocent tumours on the skin are **sebaceous cysts, angiomata** and **fibromata,** the last in the form of soft warts. The ordinary rules are applicable to their diagnosis (fig. 153). Primary **sarcomata** of the skin usually start in pigmented or in non-pigmented warts (fig. 155). The chief signs of malignancy are sudden rapidity of growth, hardening of consistence, and bleeding on slight provocation. Every tumour of the skin which has not existed for a long time, and which feels hard should be suspected of malignancy.

Slowly growing sarcomata rarely occur on the skin, and then they may be more or less pendulous (fig. 154), but never so much as lipomata. Their firm consistence, from the beginning, excludes all doubt.

Multiple sarcomatosis of the skin presents a typical picture, but fortunately the condition is rare (fig. 156).

Lipomata, which are so frequent on the back (fig. 150), have their seat of origin in the *subcutaneous fat.* They are at once identified by their lobulated form, and by the slight puckering of the overlying skin. There is no difficulty in distinguishing them from cold abscesses.

The back, just like the neck, may present symmetrical lipomata (fig. 151), as well as the more usual unilateral variety. They are generally associated with a development of multiple lipomata over the whole body, and constitute a feature of so-called Dercum's disease (see Neck).

Tubercular abscesses of the scapula often imitate perfectly the ordinary lipomata of the back, and are easily mistaken for them. They may be even more easily confused with the rare *cystic* lymphangiomata of the subcutaneous tissue. The latter, which are always of congenital origin, although they may not appear until later in life, often occur in the vicinity of the axilla. They feel soft like lipomata, but in some parts, where comparatively large cysts are present, there may be genuine fluctuation. But they are distinguished from lipomata by the fact that they are not clearly defined from the surface on which they rest, as they send processes downwards between the muscles. Sometimes the overlying skin is so thin that the whole structure is almost transparent, like a hydrocele over which the skin is made tense.

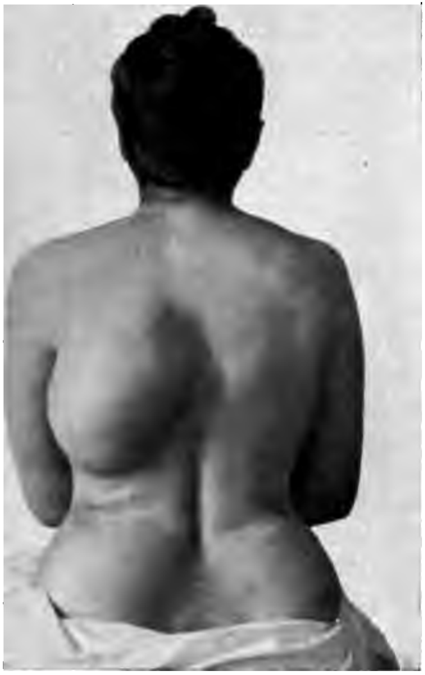


FIG. 150.—Unilateral lipoma of the back.

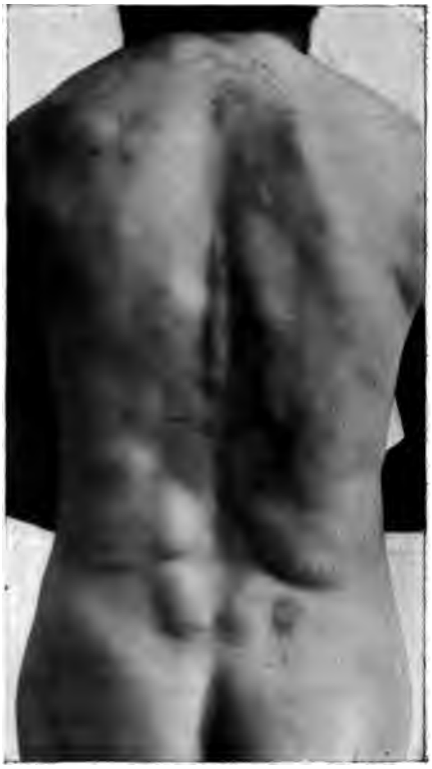


FIG. 151.—Symmetrical lipomata of the shoulders and loins.

Tumours may also arise from the *muscles* or *fasciæ*. They are mostly **sarcomata**, and more rarely **fibromata** or **lipomata**. One example will suffice.

A little boy had on his back, near the spine, a flat, long, oval tumour (fig. 152) with a lobular outline which suggested a lipoma. But it did not actually lie in the skin, which could be easily picked up over it. On the other hand, it was not connected with the bone, because it was quite movable over it. The tumour was held fast on muscular contraction, showing that it was connected with the muscles. The aponeurosis became very definitely tense over it when the muscles contracted. It was so well circumscribed that no suggestion of an infiltrating malignant growth could be entertained. Its cake-like flatness pointed to an innocent growth, and this view was more consistent with the anatomical conditions, especially the pressure of the fascia, than would be the idea of a sarcoma. The diagnosis, therefore, appeared to be : sub-aponeurotic or intra-muscular lipoma or fibroma. As a matter of fact, it was a lipoma rich in connective tissue, which had been flattened out between the muscle and aponeurosis.

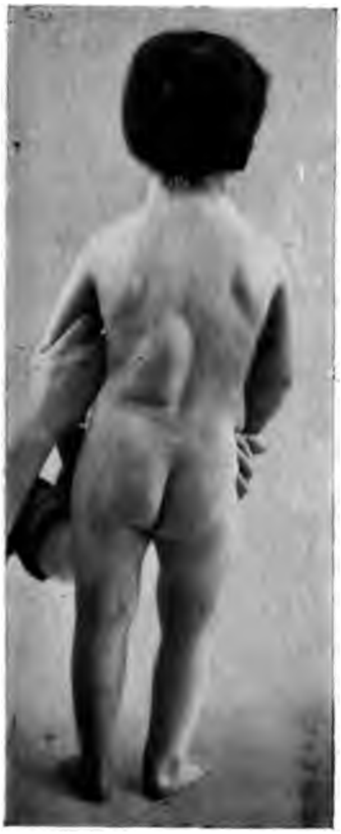


FIG. 152.—Fibro-lipoma of the muscles of the back.

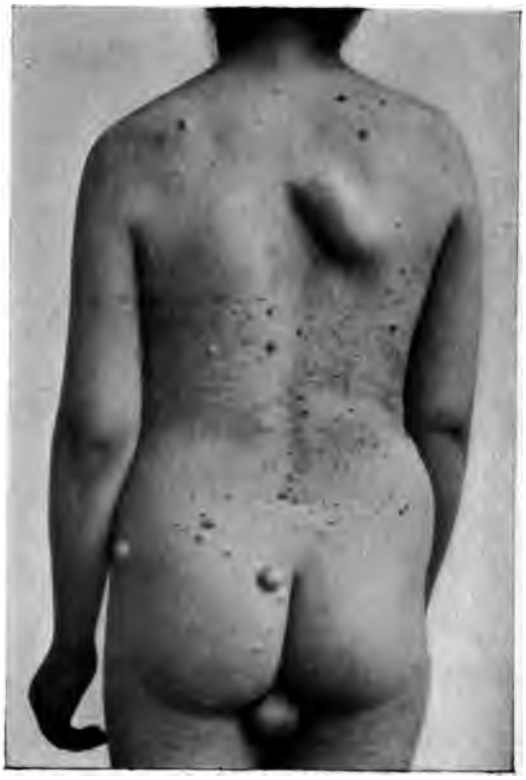


FIG. 153. — Multiple fibromata of the skin in a woman. One situated on the perinæum looks like a scrotum. Under the right scapula there is a deeply-placed neruo-fibroma

If a tumour is not movable over the *bone*, it has either arisen therefrom or become adherent to it secondarily. We assume the former if this immovability has been noted early or from the very beginning.

Primary tumours of the bone are either **enchondromata** or **sarcomata**; and much more rarely **osteomata.** Histologically, the former are innocent, but their clinical behaviour manifests all transitions to pronounced malignancy. They appear as round protuberant growths, and are distinguished by the enormous circumference which they may attain. Sarcomata may also reach to a considerable circumference. It is important to know whether the growths extend into the chest, and how far. These tumours often resemble icebergs in the respect that the portion visible is the smallest part of their mass. Auscultation and percussion may yield definite information on this point. But a skiagram is more conclusive, and one ought to be taken before venturing upon any rash removal of such a growth.

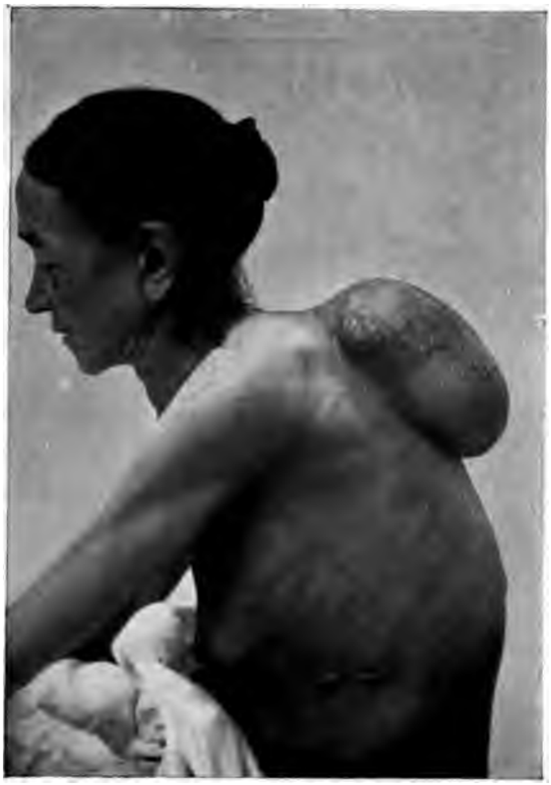


FIG. 154.—Fibro-sarcoma of the skin of the back.

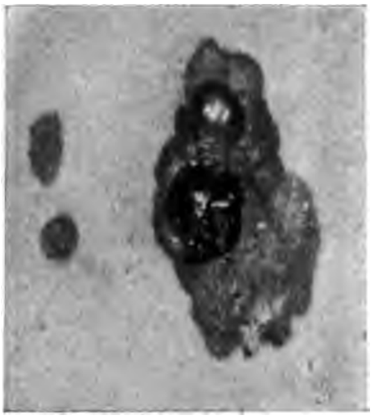


FIG. 155.—Pigmented nævus of the lumbar region, with partial sarcomatous degeneration.

Finally, if we find in the middle line of the back a tumour only slightly movable over the spine, we should at once think of **spina bifida** and its sequelæ, which we will refer to later on in detail.

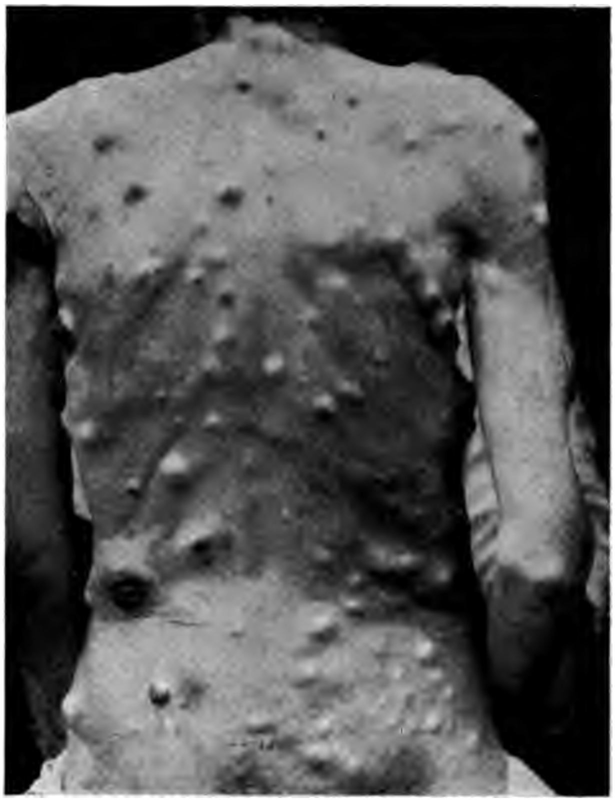


FIG. 156.—Multiple sarcomatosis of the skin.

CHAPTER XXXII.

INFLAMMATORY DISEASES OF THE BREAST.

EVERYONE feels capable of diagnosing such a superficial and easily recognizable disease as mastitis. Nevertheless it is sometimes mistaken for cancer—not only tubercular mastitis, but even the ordinary acute infective condition. The following considerations should, however, prevent error :—

(1) Tumours rarely occur before 20, and they are not very frequent between 20 and 30. On the other hand, inflammatory conditions are rare after 50.

I have, however, opened a large retro-mammary abscess, diagnosed as a new growth, in a woman of 60. The breast stood forth in a semi-spherical shape, and the swelling felt hard like a growth, because the abscess was behind the gland. Even intra-mammary subacute abscesses in old women are mistaken for carcinomata, an instance of which is depicted in fig. 157. The patient was 49 years of age, and the swelling in the breast was only slightly tender on pressure; but the œdematous indurated condition of the skin over it enabled a diagnosis to be made quite easily.

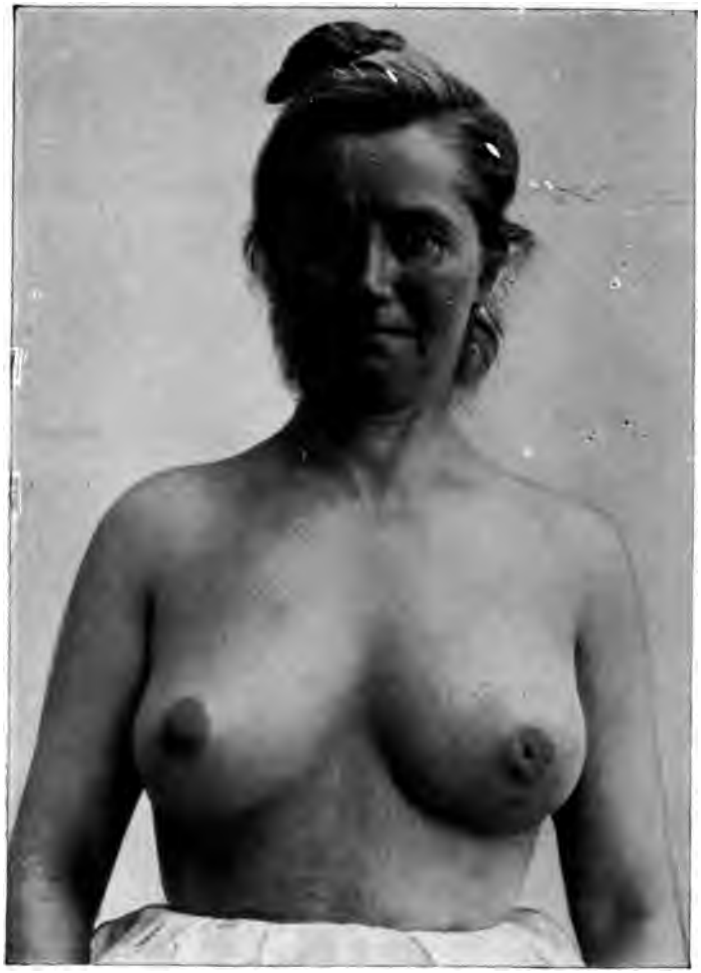


FIG. 157.—Abscess in the outer half of the left breast. Retraction, but no elevation of the nipple.

(2) An association with the *puerperal period* is of special importance for the diagnosis, because this always points to inflammation, notwithstanding the slow progress of the swelling and the absence of fever.

A case was referred to me, with an induration of the breast, which came on gradually several months after the patient had recovered from puerperal mastitis. It was considered to be suspicious of carcinoma, but a small incision cleared up the matter, by the appearance of a small amount of staphylococcic pus. In these atypical cases of mastitis sugar is sometimes found in the urine.

(3) On the other hand, it must be remembered that a rapidly growing malignant tumour with extensive destruction of tissue may cause œdema and redness of the skin before ulceration or bacterial invasion occurs, and this may lead to errors of diagnosis. But in these cases the history will usually show that a growth, independent of the skin, was present for some time before the inflammatory symptoms.

(1) ACUTE INFLAMMATIONS.

We will now consider the various forms of **mastitis,** beginning with the **acute** forms.

(*a*) The inflammation of the breast which occurs in **infancy** or at **puberty,** but sometimes also in the intervening period, in both sexes, and which very rarely suppurates, presents no difficulty in diagnosis. The gland feels like a hard round plate which is movable over the underlying muscle. It is very painful at first, and may remain tender for a long time after the subsidence of the acute symptoms.

This persistence of pain caused me to shell out the breast of a young man, at his urgent request, retaining the integument of skin and the nipple—an operation which is not justifiable in a female. An irritable condition of breast in girls at puberty has been attributed to masturbation—whether with justice remains doubtful—and this must not be confused with a bacterial inflammation.

(*b*) **Puerperal mastitis** is the type of *acute inflammation of the breast,* and in its classical form is incapable of leading to an error in diagnosis. A mistake can only arise, as previously mentioned, when the onset of the inflammation is delayed until months after the confinement, and does not occur within the first few weeks as is generally the case. But in such a case there will have been slight inflammatory attacks soon after the confinement.

The condition previously termed *stagnation of milk* is now known to be a mild infective process taking place, either in the milk congested within the ducts and their ramifications, or, as is more usual, in the connective tissue.

It is important to recognize the degree of the inflammation and the *site* of the *suppuration* for the purpose of treatment. If the temperature subsides after the initial rigor and the pain ceases after a few days, suppuration is improbable. But if slight fever persists, and if a soft area, however small, appears in the middle of the infiltrated segment of the breast, it is quite certain that pus is present.

As far as the *position* of the pus is concerned, we must distinguish between abscesses *in front* of, *in*, and *behind* the breast. The *superficial*, purely subcutaneous abscesses are usually situated in the vicinity of the areola and arise from a circumscribed superficial lymphangitis. There is no difficulty about their diagnosis.

Abscesses within the *parenchyma* appear at first as more or less well defined firm nodules, over which the skin is still normal. If spontaneous resolution does not occur, the skin becomes immovable, œdematous, and finally reddened, a soft area developing in the middle of the hard portion. If the abscess is not opened at this juncture it spreads further under the skin and pronounced fluctuation can be detected.

Deep, *retro-mammary* abscesses arise from deep intra-mammary foci, which take the nearest course and spread towards the loose connective tissue behind the breast. The whole gland may be diffusely tender, but sometimes there is a complete absence of pain and the diagnosis must be based on apparent enlargement of the breast, due to its abnormal *prominence*.

(*c*) An acute mastitis may occur at any age without the above-mentioned causes, through infection of a nipple which has been mechanically irritated, but this is rare.

(2) CHRONIC INFLAMMATIONS.

These are generally due to *tubercle*, rarely to *gumma* or *actinomycosis*. But one may repeat here what was said at the beginning of the chapter, that the sub-acute or chronic *staphylococcic* or *streptomycotic* infections also occur.

(*a*) **Tuberculosis of the breast** may appear as an *isolated nodule* and thus be mistaken for carcinoma. But the presence of other tubercular stigmata, and enlarged glands in the axilla, which rapidly become adherent to the skin and soften, would be strongly suggestive of tubercle. The diagnosis is easier if several tubercular foci are present in the breast, and especially if some of these foci have softened in places, contracted adhesions to the skin, which has become reddened and broken down (fig. 158). In the latter case the diagnosis can be confirmed by bacteriological examination of the pus and histological investigation of a piece of the granulation tissue.

It does not always follow that a cold abscess behind or near the breast really originates therein. As a matter of fact, it more frequently arises from a rib. If the lesion of the rib is not directly accessible to examination we must depend upon the absence of any change in the breast itself to suggest caries of the rib. The final verdict must, however, rest with a skiagram, which may often reveal the lesion of the rib quite distinctly.

(*b*) Tuberculosis of the breast is somewhat similar to **actinomycosis,** a few cases of which have been recorded. In the latter, however, the glands are not enlarged, and the hard infiltration is very distinctive. If an abscess or a sinus be present we must search for the characteristic granules.

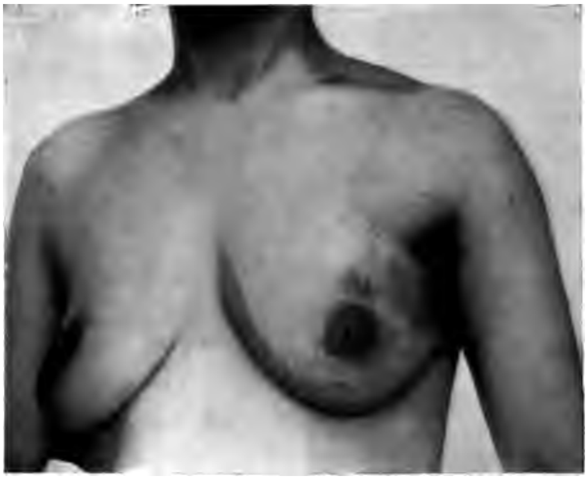


FIG. 158.—Tuberculosis of breast.

The diagnosis of **gumma** of the breast can only be arrived at by exclusion; and is confirmed by the previous history, the serum test, and the result of specific treatment.

Very rarely chronic inflammatory processes which do not suppurate, and which cannot be referred to any of the causes just mentioned, occur in the breast. *Chronic cystic mastitis*, which we will discuss in connection with tumours, is *not* of inflammatory origin.

CHAPTER XXXIII.

TUMOURS AND ALLIED STRUCTURES IN THE BREAST.

THE breast, like other glandular organs, is often the seat of structures which are not genuine tumours, but which are not sharply differentiated from them. They have nothing to do with bacterial inflammation. As the epithelium and connective tissue are both concerned in these processes they are best designated by the general term of fibro-epithelial degeneration. Examples of this morbid process occur in the *thyroid as goitre*, and in the *prostate as hypertrophy*. In the *breast* the following types may be differentiated :—

(*a*) Preponderating cyst formation, either as a solitary cyst or as a conglomeration of small cysts (Reclus' or König's disease).

(*b*) Preponderating proliferation of the connective tissue; formation of so-called fibro-adenoma phyllodes.

(*c*) Preponderating proliferation of the epithelium: Fibro-adenomata of purely adenomatous or of papillary character.

All these changes may occur, either in a diffuse form or as circumscribed encapsulated tumours. They may occur as single or as multiple nodules, and are frequently present in both breasts. Sometimes all the various types are combined in one growth; at others, nodules which have arisen at the same time in both breasts may manifest quite different characters. Very frequently, as one might expect, *cancerous* or *sarcomatous* degeneration occurs. We have digressed somewhat into these pathological details, but in view of the existing controversy on the matter this was not superfluous. We will now proceed with the diagnosis proper of tumours of the breast.

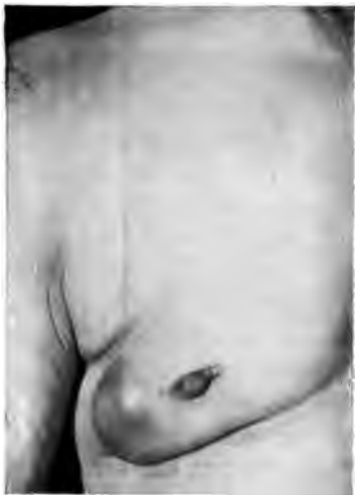


FIG. 159.—Superficial cyst of the breast.

If we have decided that a swelling of the breast is not of inflammatory or infective origin, and therefore regard it as the result of *fibro-epithelial degeneration*, or as a genuine *new growth*, we are met with the great question of **innocence** or **malignancy**—in other words, is the case one for immediate operation, or is it one wherein the advisability of operation may be discussed with the patient? The precise histology of the disease is of very secondary importance in comparison with this question of innocence or malignancy. The first point to ascertain is whether the tumour is single or multiple.

A.—MULTIPLE TUMOURS.

If the tumours have arisen in *both* breasts about the same time, or if there are several in the *same* breast, we may conclude with great probability that they represent the innocent process of fibro-epithelial degeneration. But in order to establish the diagnosis they must possess the characteristics to be described in the following section. It is, however, quite possible for one nodule of an originally innocent fibro-adenoma to became cancerous, or cancer may suddenly burst forth in any old harmless fibro-adenoma. Unfortunately, both patients and practitioners are liable to forget this possibility, and many hesitate to make the diagnosis of cancer because some old cysts are present, either on the same or the opposite breast.

B.—SINGLE TUMOURS.

We will divide these according to their size, because the questions which arise in regard to them vary according to their dimensions.

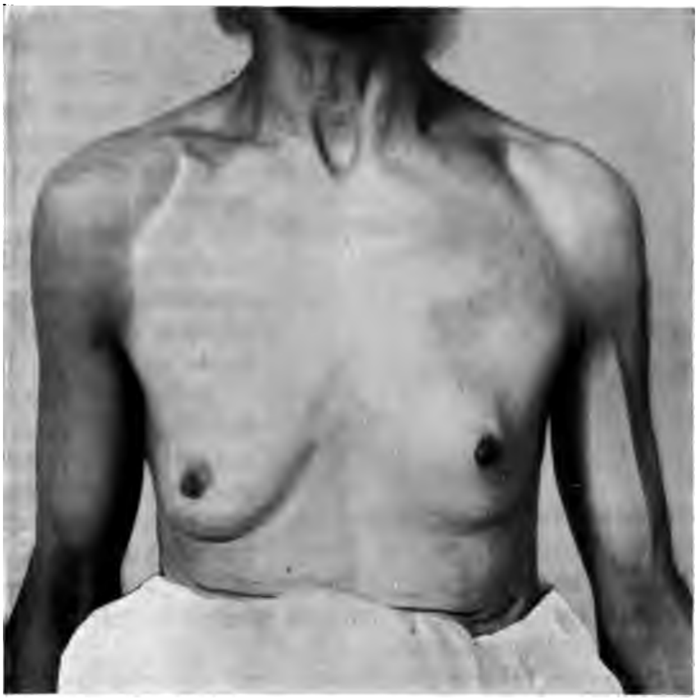


FIG. 160.—Early scirrhus. Nipple drawn up and slightly retracted.

(1) SMALL AND MEDIUM-SIZED TUMOURS.

We begin with *small and medium-sized tumours*, *i.e.*, those which do not exceed a fist in size.

The fundamental sign which after a little palpation almost always differentiates between innocence and malignancy is the *movability of the tumour in relation to the rest of the breast tissue.* A certain amount of practice is required to estimate this mobility correctly, but it can be acquired by the attentive examination of a few cases. An index finger is placed on each of two points on opposite sides of the tumour, which is jerked backwards and forwards between them, a shaking movement being imparted to it at the same time. If the tumour yields easily to this manœuvre it is innocent. If this mobility is absent it is practically conclusive of malignancy, even if the growth is not adherent to the skin or to the pectoral fascia, and the nipple does not yet show any indication of being drawn in. Only an inflammatory attack can temporarily deprive an innocent tumour (cyst) of its mobility. If pressure is made on the tumour with the hand flat upon the chest it is less distinctly felt if it is a cyst, whereas if it is a cancer it is more distinctly felt. Whenever I have been in doubt about the degree of mobility, I have generally found a malignant tumour at the operation.

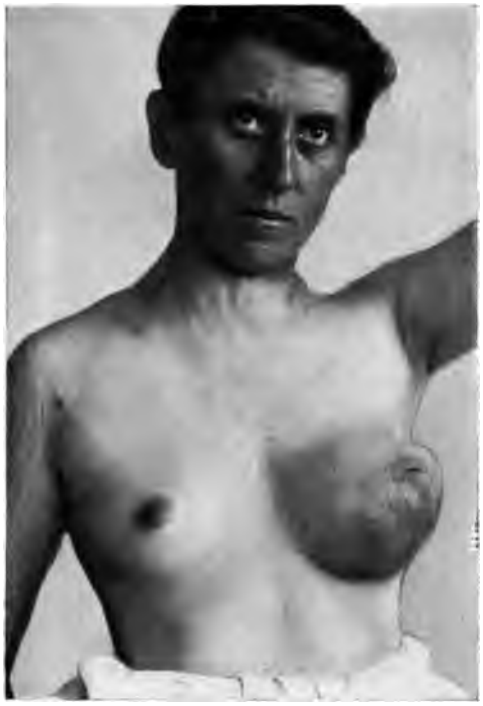


FIG. 161.—Polycystic fibro-adenoma.

Our decision must never be influenced by the patient. Every surgeon knows of cases wherein the apprehensive patient considered her harmless cyst to be a cancer, and also of cases wherein a "hardening" of the breast has been shown casually to the medical attendant, when it had already exceeded the limits of cure by operation.

(*a*) Having decided from the above considerations that the tumour is **innocent,** we may now proceed to determine to which of the foregoing anatomico-pathological groups it should be ascribed. If the breast is diffusely indurated over a large extent, that is to say, if it has become converted into a conglomeration of small hard movable nodules, the case is one of the formation of numerous little *cysts.*

Single nodules up to the size of a goose's egg are either *encapsuled fibro-adenomata* or *solitary cysts.* As the latter do not show any fluctuation owing to their extreme tension, the clinical differentiation is often impossible.

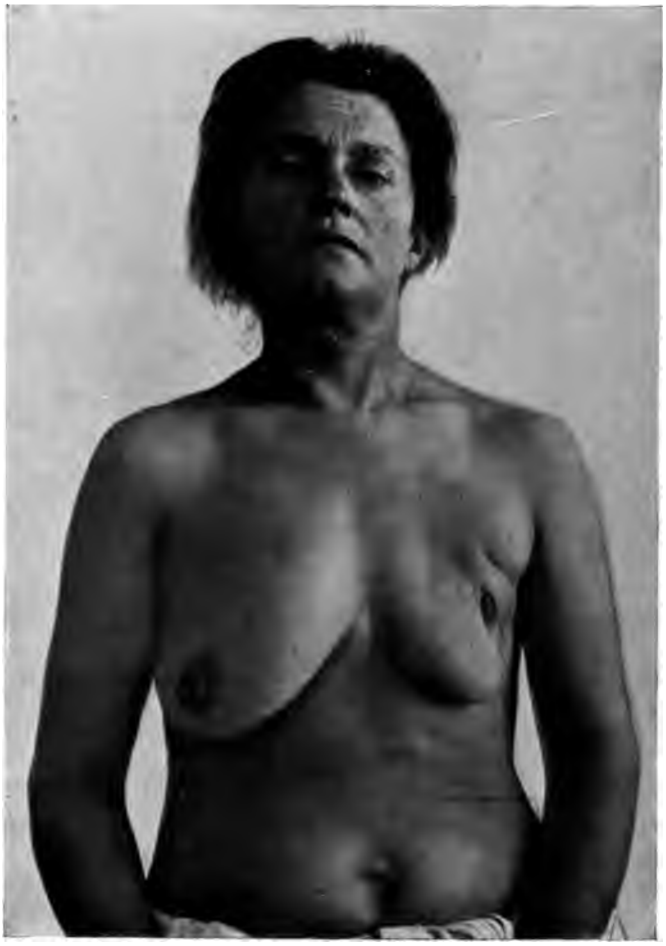


Fig. 162.—Advanced scirrhus with retraction and elevation of nipple and diminution in size of its areola.

Cysts are usually indicated by variation in volume, by increase in size and by pains during menstruation or at the onset of pregnancy, and by the discharge of milky fluid from the nipple. A brownish or sanious secretion especially signifies a papillary cyst, or an adeno-papilloma developed within a cyst, which stands just on the border line of malignancy (so-called "bleeding breast," see below). Superficial cysts have a bluish transparency and fluctuation is often present (fig. 159).

If the nodule exceeds a goose's egg in size the case is not one of a solitary cyst, but is some form of encapsuled fibro-adenoma, or the whole breast may have become changed into a mass of multiple cysts (fig. 161).

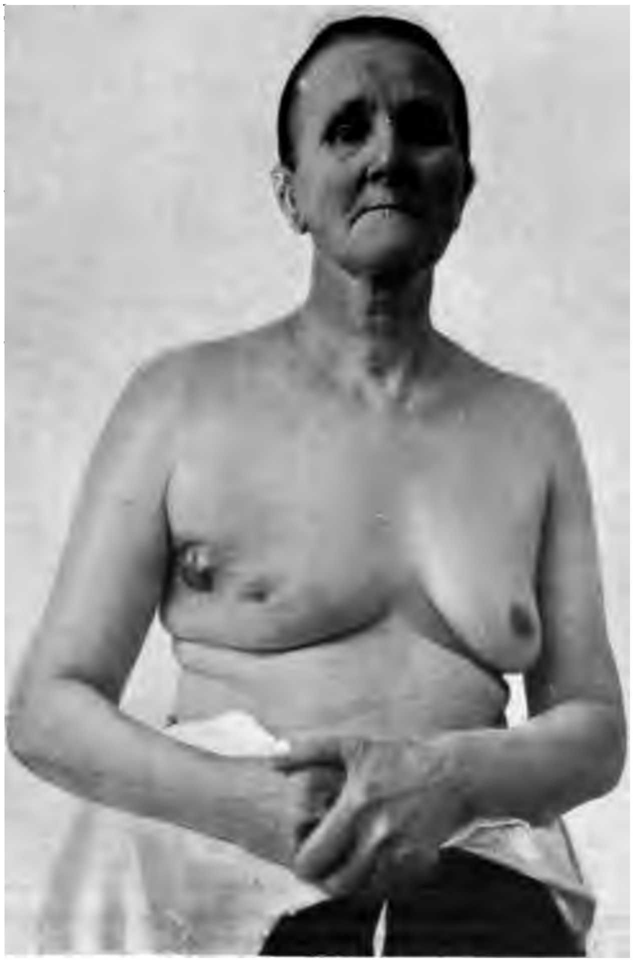


FIG. 163.—Cancer of breast, slightly contracting, situated at the periphery. Elevation and retraction of nipple, diminution of areola.

We have hitherto regarded the fact that a tumour is freely movable over the rest of the breast tissue as a positive sign of innocence. But, unfortunately, there are exceptions. A primary cancer occasionally remains quite movable for a long time; but the history is distinctive in such a case. If the tumour has only been present a matter of months it is probably cancer; if for a year or more it is a fibro-adenoma. Further, fibro-adenomata not rarely undergo cancerous change, and this alteration is not signified by any recognizable clinical symptom. For this reason we should propose to the patient the removal of any circumscribed tumour, however innocent it may appear to be. If she agrees, the growth is removed while the conditions are healthy, or better still the whole breast is shelled out.

The excision of a small piece of a mammary tumour for diagnosis is of no value, for the piece removed may happen to come from a portion which is harmless, whereas a cancerous portion may still be present. The following case indicates the difficulty of diagnosis :—

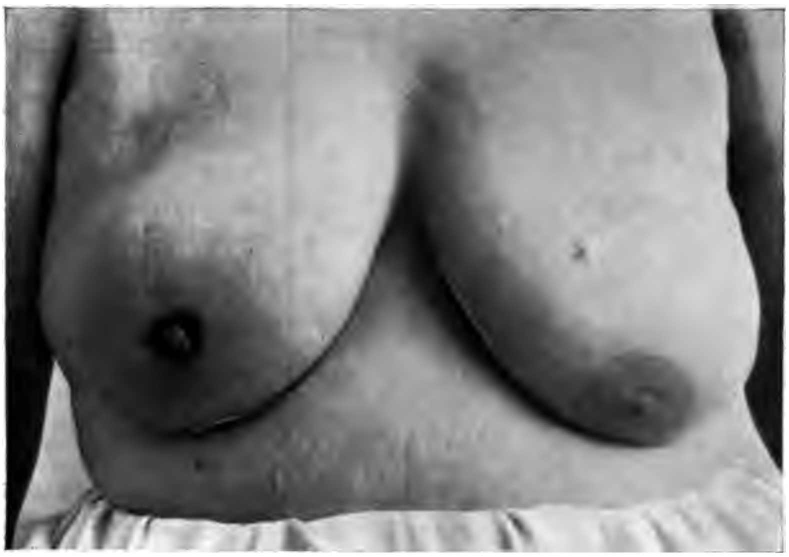


FIG. 164.—Ulcerated cancer of right breast in patient aged 65. Apparently a cutaneous cancer of the areola (with ulceration above and to inner side of nipple), but really a contracting cancer of the breast with elevation of nipple and diminution of the areola.

A person, aged 50, noticed a small movable lump in each breast. They both felt alike, were diagnosed as cysts and removed. Histological examination showed that the tumour on the right side was an early fibro-adenoma, and that the one on the left side was an innocent cyst. The latter preparation was, however, examined again some years later in detail, and a circumscribed, classical infiltrating carcinoma a few millimetres in size was noted close to the cyst. Meanwhile, enlarged glands appeared in both axillæ, and after their removal it was shown that they were—tubercular. The patient came from a tubercular stock.

(*b*) If the tumour is only slightly movable or quite immovable over the rest of the breast tissue, our diagnosis must be a **malignant** growth, especially a **carcinoma,** even if the ordinary criteria of this disease are absent, viz., retraction and elevation of the nipple, enlarged glands in the axilla, and adhesions to the skin and pectoral muscles. All these signs eventually make their appearance, but the practitioner who awaits them before making a diagnosis is in no enviable position.

Retraction of the nipple is an early sign only in the contracting forms of cancer ; but it sometimes occurs in chronic cystic mastitis and even in chronic abscesses. The *diminution of the areola around the nipple* is much more distinctive of cancer (fig. 162), and the elevation of the nipple is of great significance (figs. 160-164) because it indicates a process of contraction and points to cancer, even if the nipple itself is scarcely retracted at all (fig. 160).

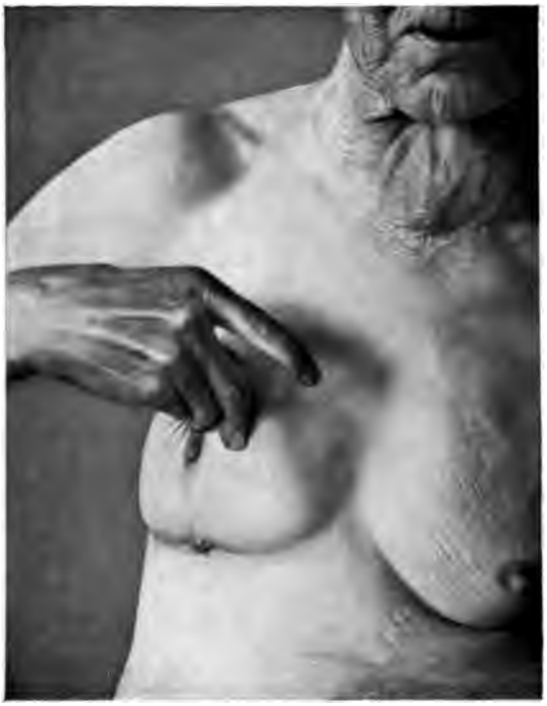


FIG. 165.—Small contracting cancer in the fold under the breast.

Enlargement of the axillary glands occurs in some slight degree even in the case of innocent cysts, and nearly always in tubercular disease. Tubercular glands are usually softer than the malignant variety and break through the skin sooner. Sarcoma attacks the glands much more rarely than carcinoma. The difficulty of detecting early cancerous enlargement of the glands, in fat women, is frequently realized first at the operation. But the *demonstration of cancerous glands has a prognostic rather than a diagnostic importance ; cancer of the breast can and must be recognized without them.*

Exceptionally, the enlargement of the glands dominates the whole clinical picture and the cancer itself is overlooked. Thus, a patient, aged 47, was treated for eighteen months for enlarged movable glands in the left axilla, which, according to the family history, might have been tubercular. This diagnosis was apparently supported by the occurrence of similar glands in the right side of the neck.

Careful examination, however, showed that there was on the left side a very slight induration behind the nipple, which had hitherto been overlooked. Histological examination of the breast and glands confirmed the diagnosis of cancer.

Adhesion to the pectoral muscles is recognized by the fact that the tumour is movable when the muscle is relaxed, and that it appears to be immovable when the muscle is contracted.

We have not yet said anything about *ulceration*, because this does not afford any fresh materal for diagnosis. It occurs in those forms which have a tendency to rapid destruction and wherein the whole tumour resembles an ulcer (fig. 167), and also in the scirrhous variety wherein the skin is soon involved, especially when situated near the nipple (fig. 164), or in the fold of skin at the lower border of the breast (fig. 165). Finally, almost every form of carcinoma will naturally ulcerate, if left long enough.

The small contracting cancers of the nipple (fig. 164), and those which occur in the fold of skin under the breast (fig. 165), represent typical forms. Their early ulceration and their whole appearance lead the beginner to the diagnosis of cancroid. They are really cancers of the breast with portions deeply situated, and on palpation they are recognized to be much larger than their superficial appearance suggests.

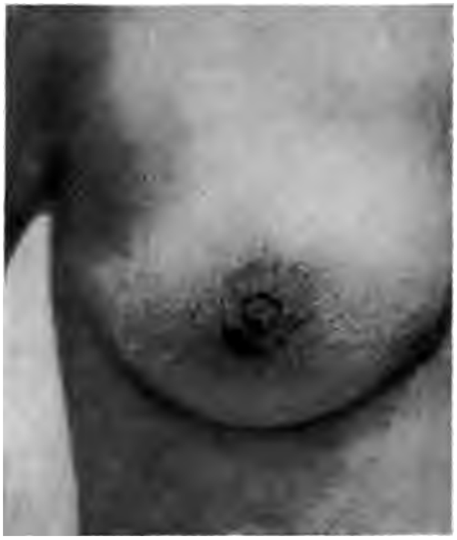


FIG. 166.—Primary chancre of nipple.

The former variety may be confused with primary chancre of the nipple—a condition which has also been observed in the male sex.

If an eczematous condition of the nipple and its vicinity has preceded the development of the tumour we diagnose the well-known form of cancer, termed *Paget's disease*. Every obstinate eczema in this region must be suspected, although there may be no tumour present.

The only constant sign of cancer common to all forms is its *slight mobility compared with the healthy breast*. All the other characteristics are variable, and there are numerous transitions from the soft medullary cancer rich in cells, which frequently attains the size of a fist and more (fig. 168), to the contracting scirrhus, poor in cells, which constitutes a loss of tissue, although it is actually a growth (figs. 160 and 162).

Having decided upon the diagnosis of cancer, the matter of accurate *prognosis* claims our attention. If *secondary* growths are found futher discussion is useless. In this connection the *vertebral column* should be thought of, and it is worth while examining the breast in cases of unexplained sciatica or intercostal neuralgia.

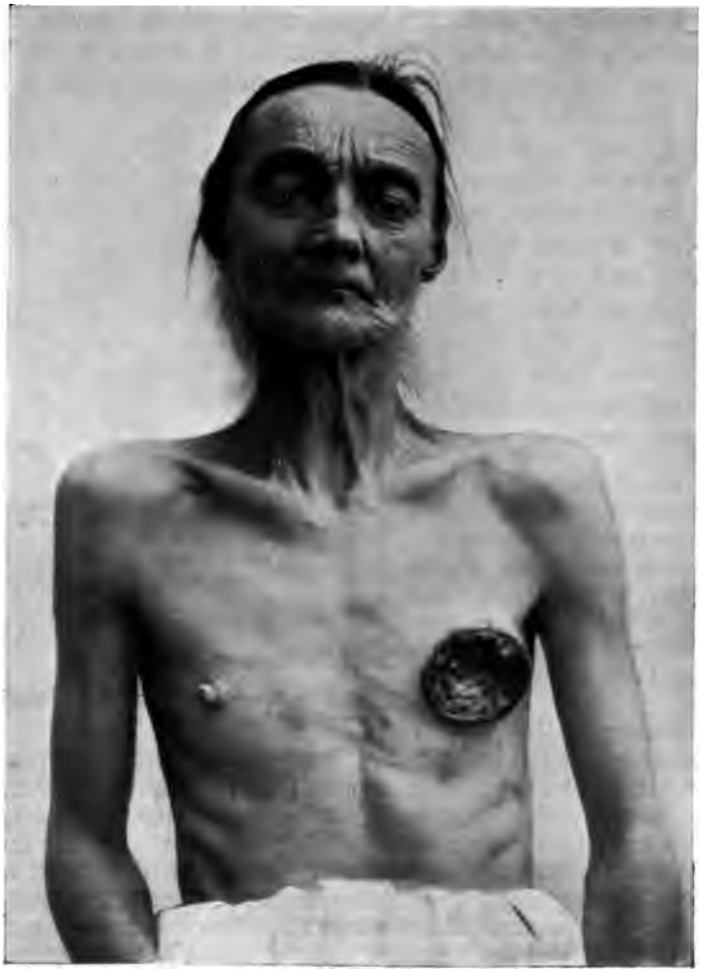


FIG. 167.—A rapidly breaking-down cancer of breast. The whole tumour converted into an ulcer.

A patient was undergoing electrical treatment for several months by a neurologist for lumbar neuralgia. Notwithstanding the sparks, there was no illumination of the breast until the cancer therein became an inoperable scirrhus, which was accidentally discovered by the family practitioner. Paraplegia soon set in.

There are three other signs which exclude the possibility of a complete recovery, and these must be considered before any advice is offered: (1) *adhesion to the ribs;* (2) the presence of *scattered cancerous nodules in the surrounding skin* (fig. 169); and (3) *involvement of the supra-clavicular glands.* If there are any indications for an operation in these circumstances it can only be one for relief of local symptoms.

We have hitherto limited our discussion of malignant growths to cancer, and have said nothing about **sarcomata.** If these are of an *infiltrating* type from the commencement they are indistinguishable clinically from medullary cancer. All that we have said about the latter applies to this form of sarcoma, with the exception of the involvement of the lymphatic glands. *Encapsuled* sarcomata, as long as they do not exceed a fist in size, are only distinguishable from fibro-adenomata by their more rapid growth. The diagnosis of sarcoma is of more importance in regard to the larger tumours of the breast, to which we will now turn.

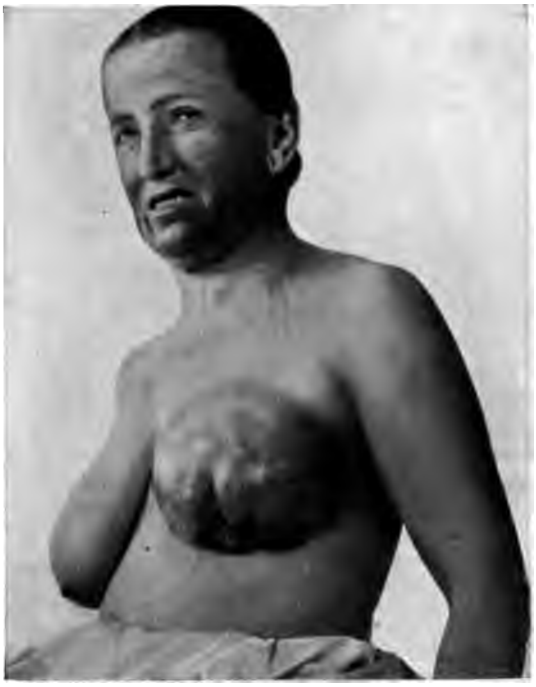


FIG. 168.—Medullary cancer of breast.

(2) LARGE TUMOURS.

A *large* new growth of the breast may be merely an example of hypertrophy (*giant growth*), and then it is usually bilateral. Apart from this we have to distinguish those that belong to the class of fibro-adenomata on the one hand—*i.e.*, **fibro-adenoma** or **cysto-adenoma** and **cysto-sarcoma** phyllodes—from **true sarcomata** on the other hand. Notwithstanding the modern zeal for operation both classes are still responsible for *enormous tumours.*

Thus a patient allowed her tumour (fig. 170) to attain the weight of 5½ kilos and to become extensively putrefied in the course of a year during which she was under "nature treatment." That the patient eventually became intensely cachectic was not a matter of surprise considering the amount of putrefaction and the dietetic restrictions which she underwent. Nor was it at all surprising that the "nature-curer" claimed the credit for the relief which the operation gave. At the operation the growth was still well encapsuled, and histologically it was composed of a typical fibro-adenoma with some partial sarcomatous degeneration of the stroma. Two years afterwards the patient succumbed to multiple metastases, which consisted histologically of pure spindle-celled sarcomata.

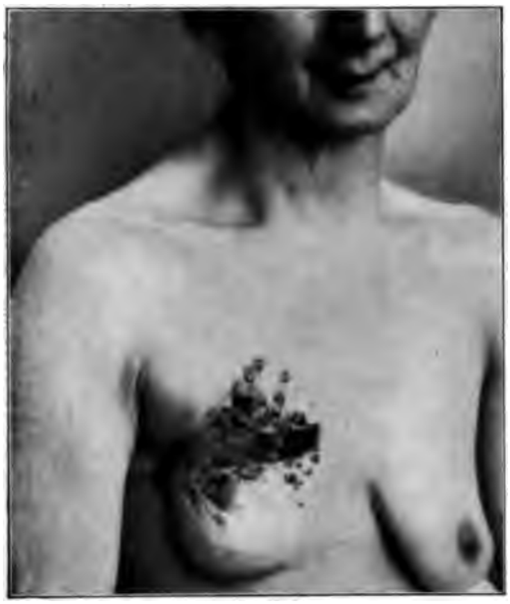


FIG. 169.—Contracting cancer of breast, with numerous cancerous nodules in the surrounding skin.

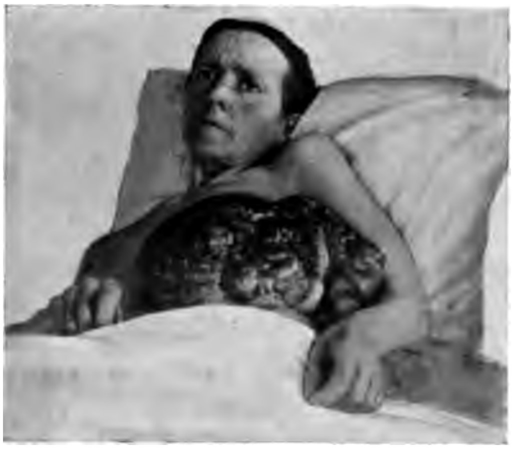


FIG. 170. — Fibro-adenoma phyllodes of breast, broken through and partially undergone sarcomatous degeneration.

Fibro-adenomata or cysto-adenomata phyllodes, as well as the sarcomatous degeneration of the latter, known as cysto-sarcomata phyllodes, are well encapsuled tumours, which only ulcerate if the skin over them becomes tightly stretched. Metastases occur in the sarcomatous form, but more rarely than in the case of *primary sarcoma* of the breast.

The latter are either encapsuled or infiltrating, and occur under the most varied histological forms. Cystic formations also take place in these cases, but not as the product of epithelial structures as in cysto-sarcoma proper, but as the result of tissue necrosis.

If the tumour shows no infiltrating growth the differential diagnosis must be based on the history. If it has been present for a year or more, and has been growing regularly or by fits and starts, the tumour is a fibro-adenoma. The sudden rapid growth of a tumour which has been quiet for years indicates sarcomatous degeneration. A tumour of wide extent, despite short duration, is a primary sarcoma.

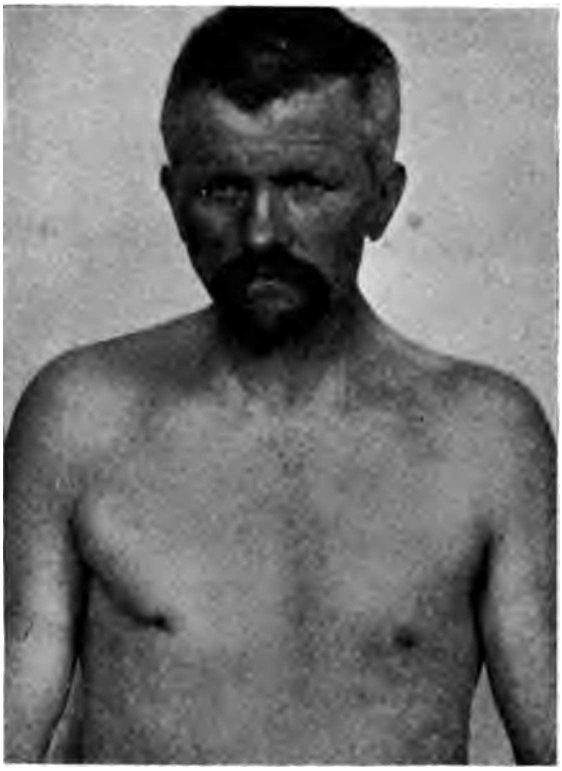


FIG. 171.—Scirrhus of breast in a man.

A word must here be said about the "**bleeding breast.**" The exudation of fluid, varying in colour from brownish-red to pure blood-red, either spontaneously or on pressure, is usually a sign of fibro-epithelial degeneration. It occurs especially when papillomatous proliferations are present in cysts, communicating with the milk ducts. The symptom is, therefore, not so serious as the patient usually imagines; but she should not be reassured too confidently, for I have also seen this bleeding in cancer.

In this case, however, there were some fibro-epithelial changes, innocent cysts, from which the bleeding obviously emanated, in addition to the malignant portion of the growth. But the history indicated that the hæmorrhages occurred with the onset of the carcinoma, which was still recent. It follows, therefore, that the carcinoma must have indirectly excited the bleeding.

As examples of purely innocent tumours of the breast, there should be mentioned lipoma, which is rare, and chondroma, which is still more rare. The former usually occurs *near* and not *in* the breast, and is recognized by its softness, just as the latter is recognized by its cartilaginous hardness. The whole breast seems to be enlarged in lipoma, but it is in a dependent position, just like the healthy breast. Finally, it should be stated that the male breast may be affected with any of these forms of tumour, but, according to Schuchart, in the proportion of 1 to 100. An example is illustrated in fig. 171.

PART IV.

SURGICAL DISEASES OF THE ABDOMINAL AND PELVIC VISCERA.

CHAPTER XXXIV.

DISPLACEMENTS OF THE ABDOMINAL VISCERA.

THE accuracy of the topographical diagnosis of abdominal diseases depends upon the assumption that the viscera occupy their normal position. But this is not always the case. All the abdominal viscera are liable, in more or less degree, to displacements, some of which date from birth, while others are acquired in later life.

A.—We may begin with the **congenital displacements.** The only paired viscera in the abdominal cavity—the kidneys—are occasionally subject to peculiar conditions.

The kidneys, for example, may be fused together and lie in front of the spine, in the shape of a horse shoe or a cake. They may also be fused together at their extremities and lie on the same side, one above the other. If one kidney is entirely absent, the other is abnormally large. Congenital displacement of one or both kidneys into the pelvis is of still greater importance diagnostically. Sometimes the displaced kidney lies at the side, sometimes it is in the middle; occasionally it is found in the false pelvis, at other times even in the true pelvis. With the methods accessible to the practitioner, it is impossible to diagnose these anomalies with any certainty; more especially, as such kidneys, do not exhibit the mobility of the ordinary movable kidneys, nor are they capable of being replaced into their normal position. Therefore in operating upon a "tumour of the adnexa," the diagnosis of which is not clear, it is always desirable to think of the possibility of this condition. The significance of a mistake in connection with a displaced *solitary kidney* need not be mentioned.

These cases can only be clearly distinguished by skiagraphy combined with catheterism of the ureters, or, better still, by a skiagram taken after the renal pelvis has been filled wlth collargol.

The fundamental variety of displacement of the *unpaired* abdominal organs is the "typus inversus." This is comparatively easily detected on clinical examination, provided the thoracic viscera are affected in the like manner. But if the displacement only concerns the abdominal organs, it is very likely to be overlooked, although palpation and percussion would probably reveal the rare cases wherein the positions of the **liver** and **spleen** are reversed. A doubtful case is immediately cleared up by a skiagram, because the position of the stomach is also reversed in these circumstances.

Displacement of the *intestine alone* is much more frequent and therefore of greater surgical importance.

The following are the chief varieties which have been distinguished :—

(1) The large intestine lies in its whole extent, *behind the small intestine*, because of the failure of the umbilical loop to revolve (*retroposition*). The mesentery may either be free or may contract adhesions with the posterior abdominal wall.

(2) The entire large intestine lies on the *left side of the abdomen* because, although the umbilical loop has revolved in the right direction, it has failed to do so completely, *i.e.*, to the extent of permitting decussation of the small and large intestine (*sinistro-position*). The mesentery may either be free or may have contracted secondary adhesions. In the former case both small and large intestine are connected with a free, common mesentery, the so-called *mesenterium commune*.

(3) The entire large intestine is in the *right half of the abdomen*, because the umbilical loop has incompletely revolved in the wrong direction (*dextro-position*). The condition of the mesentery is as in No. 2.

(4) There has been complete decussation of the small and large intestine, but in a *reversed position*, because, although the umbilical loop has revolved completely the direction has been wrong (*situs inversus abdominalis partialis inferior*).

These are the extreme varieties, but a much more frequent abnormality is one which we may regard as an intermediate form between the normal position and the left-sided position of the large intestine, with free mesentery. Here the cæcum and ascending colon possess a free mesentery, which merges with that of the lowest coil of the small intestine. At the same time the ascending colon is frequently shortened, so that the cæcum is abnormally high. If there is no ascending colon at all, and the cæcum lies directly against the border of the liver, we are within the border line of a left-sided position. We can tell this at a glance when the cæcum is so far displaced to the left that the large and small intestine no longer decussate. The significance of this abnormality may be gathered from the fact that it is found in its mildest form—*mesenterium commune ilio-cæcale*—in 10 per cent. of all autopsies. The more pronounced variety has certainly been encountered by all surgeons during the performance of laparotomies.

There are two matters to which practical importance attaches in connection with these displacements: (1) The *position of the appendix* and (2) the question of the *decussation of small and large intestine.*

Let us begin with the *appendix.*

Whereas this may lie in the true pelvis in cases of enteroptosis or when the cæcum is abnormally long, it may be found *high up* in front of the right kidney, at the edge of the liver, or even under the liver close to the gall bladder in cases of shortening of the ascending colon, which are so often associated with a free mesentery of the ileo-cæcal coil. I have found it in all these situations during laparotomy. The more free the ileo-cæcal coil, the nearer it will be to the middle line. When the large intestine is displaced to the left, it usually lies in the umbilical region or even to the left of it. In complete transposition it is found in the left side of the pelvic cavity. In this condition the entire position of the intestine is that of the normal, as seen in a mirror. It follows, therefore, that the appendix has made a complete circuit of the abdomen, and we must be prepared to meet with it anywhere.

The matter of the *decussation of the small and large intestine* is not so much one of diagnostic interest as of technical importance for operation. In all cases of incomplete revolving of the umbilical coil with a free mesentery this decussation is absent. This demands special notice because it is customary in performing gastro-enterostomy to search for the highest coil of small intestine, where it comes out under the transverse colon. If, on opening the abdomen, the position of the intestine shows that decussation has not taken place, we must follow the duodenum in order to find the highest coil of jejunum. In cases wherein the large intestine is on the left, the duodenum winds towards the region of the right kidney, and thence passes into the jejunum in the vicinity of the right side of the pelvis.

Besides these typical displacements, there are other rarer anomalies which do not admit of classification, and are only accidentally recognized in a skiagram or discovered during an operation. The intestinal displacements caused by diaphragmatic hernia belong to this group.

On one occasion the transverse colon was found, drawn up as far as the ensiform process and held tightly there, by means of the omentum, which was involved in a congenital diaphragmatic hernia into Morgagni's space.

Even the typical displacements previously referred to cannot be diagnosed clinically without the aid of a skiagram. We shall discuss the technique connected therewith later on.

B. **Acquired displacements** of the abdominal organs are grouped together under the term of enteroptosis, even if the displacement concerns one viscus only. French clinicians have been familiar with this condition for many years, but a due prominence has lately been given to it by Stiller. In regarding it, however, as a symptom of "asthenic constitutional disease," he merely *paraphrases* his observations and does not *explain* the pathology of the condition. We really do not possess any genuine explanation of the clinical picture presented by enteroptosis, and we will therefore not enter here into the discussion of theories. We shall only refer to the symptomology of those forms which are of diagnostic importance, leaving the clinical details to the chapters dealing with the individual organs.

Even the public are aware that the **kidney** may be movable, and **floating kidneys** which, a few decades ago, were unknown to medical men, have now become the common property of the civilized world.

Similarly but much more rarely, the **liver** and **spleen** become movable. But whereas, a **movable liver** is due to general relaxation of the suspensory ligaments, a movable spleen depends upon some morbid enlargement of the organ.

The downward displacement of the spleen is thus the only acquired visceral malposition which depends upon some morbid condition of the organ itself rather than upon weakness of its suspensory ligaments.

A **movable spleen** is easily recognized by its sharp anterior border, and by the fact that the splenic dulness is absent from its normal situation.

This sharp border serves as a guide, if the spleen is situated at a distance from its normal position. I had a case of a young lady who was sent to Europe from a malarial country on account of an "ovarian tumour." She had a tumour, occupying the *right half* of the abdomen and the *true pelvis*, which presented on the right side a remarkably sharp border. The normal splenic dulness was absent. This sufficed for the diagnosis of a movable spleen, and the heavy organ, weighing two and a half kilos, was removed. The pedicle of the spleen was drawn out over the transverse colon, and ran downwards to the right.

We must consider ptosis of the stomach and of the intestine, together, not only because of their clinical connection, but also because they are recognized by the same diagnostic methods. We shall begin with **palpation.**

It has long been known that palpation of the kidneys, liver and spleen renders definite indications for the diagnosis of acquired ptosis, but it was not appreciated that indications of ptosis of stomach and intestine might be obtained in the same way. Glénard and his school have indeed been teaching, for the last twenty-five years, how to palpate the large intestine. They erred, however, in their view that only a diseased, or at any rate a morbidly contracted large intestine is palpable. This error has been exposed by Obrastzow, who has shown that a considerable portion of the course of the large intestine, and also a certain portion of the normal stomach, can be demonstrated by palpation, even in health. This has been confirmed by all who have worked systematically at the subject. The frequency with which one section or other of intestine, or portion of stomach, can be palpated, depends not only upon the experience of the examiner, but also upon the clinical material available. Patients suffering mainly from medical diseases (Hausmann) will yield a higher percentage of positive results than those suffering from surgical affections, because the latter group will include many cases of meteorism and inflammatory diseases. The greatest care must be taken not to perform any systematic palpation of the individual portions of the intestine, if there is the slightest risk of causing any damage, *e.g.*, in all recent acute inflammations of the biliary passages, the appendix or the bowel. It is much better to remain in doubt in regard to the course of the intestine, than to burst an encapsuled abscess, or to rupture an appendix which is threatening to perforate.

What are we able to feel through a relaxed abdominal wall, which is not too fat ?

(*a*) Every portion of the digestive canal which contracts upon its contents, against an obstruction (gastric and intestinal rigidity).

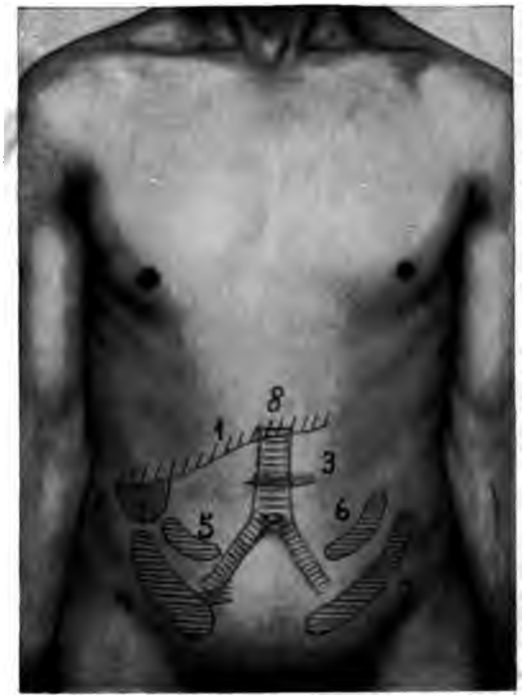


FIG. 172. — Diagrammatic representation of the abdominal organs which can most frequently be felt. (1) Liver; (2) Right kidney; (3) Greater curvature of the stomach; (4) Cæcum and ascending colon; (5) Beginning of transverse colon; (6) End of transverse colon; (7) Descending colon and upper segment of sigmoid flexure; (8) Abdominal aorta and iliac arteries.

(*b*) The large intestine, even if it contracts in an empty state (*la corde colique*) or when it is more or less filled with solid fæces.

(*c*) Any uncontracted section of bowel, which can be rolled, in a localized manner, on a firm underlying surface.

This leads us to the following conclusions as far as the various sections of the gastro-intestinal canal are concerned (see also fig. 172).

In the *stomach*, the pyloric region can be felt if it is not overlain by the liver; and the greater curvature, if it is not too low down, on account of ptosis. If the stomach has dropped considerably, the pancreas, which undergoes less displacement, may often be felt lying transversely in front of the vertebral column.

In regard to the *small intestine*, it is only possible to feel the termination of the last coil where it opens into the cæcum, even in the most favourable circumstances, as Hausmann correctly remarks.

It is very rarely possible, if at all, to feel the *appendix* in normal conditions. The termination of the final portion of the small intestine (Hausmann) or as I would suggest, at any rate its lower border, is often felt and this is assumed to be the appendix. In *pathological conditions*, a mass formed of appendix and adherent omentum, and frequently also of adjoining coils of intestine, is often taken to be the appendix.

In most patients it is possible to feel the cæcum and the ascending colon, almost as far as the hepatic flexure, and in the majority of patients the descending colon is palpable together with the upper segment of the sigmoid flexure. We are sometimes able to feel the beginning of the tranverse colon, to the inner side of the ascending colon, and its end, to the inner side of the descending colon. But the main portion of the transverse colon is only palpable when it is sufficiently high above the symphysis to permit of its being rolled on the vertebral spine, and if the patient is not fat and the small intestine only slightly filled. This combination of circumstances does not however, often occur. A transverse colon which does not lie too low, which is in a condition of spastic contraction can always be felt, but the renal flexure of the large intestine can practically never be palpated.

Although palpation affords many indications as to the position of individual portions of the bowel, the results obtained by this method do not equal in reliability those which are furnished by a **skiagram.**

A preliminary word in regard to *method*. It is simple enough as far as the stomach is concerned. Changes in position are visible immediately, upon the screen or upon the plate, if the patient is examined in an erect and recumbent posture directly after taking the appropriate shadow-forming meal (*see* under "Diseases of Stomach").

In regard to the *large intestine*, the most suitable procedure would appear to be that of injecting some shadow-forming, thin fluid emulsion, on the same principle as the old practice of distending the rectum with gas, or filling it with water—and this method has been largely employed. But it is becoming quite clear that the filling of portions of the bowel, which are intended to hold rather solid contents, with large quantities of liquid, is not suitable for revealing the correct position of the large intestine (*see* figs 173 and 174). A better conception of the position of the large intestine can be obtained by giving the shadow-forming meal by the stomach, and then following its progress through the intestine by means of screen examinations or plate impressions. It is as a rule sufficient to take impressions after six to eight hours, and after twenty-four hours, and again after forty-eight hours if there is any unusual sluggishness of intestinal movements, in order to draw a correct conclusion as to the position of the whole large intestine. If we wish to confirm the result by a bismuth enema, the amount injected must not exceed 1 to 1½ litres.

The X-rays examinations have clearly pointed out what the essential feature is, in the diagnosis of visceral displacements. Thus, in the *stomach* neither a low-lying greater curvature, nor the position of the smaller curvature, which usually runs in a vertical direction, is of so much importance as the position of the pylorus. A flabby weak stomach may become temporarily distended to a low level from the pressure of its semi-solid contents, without the pylorus necessarily having been dragged down. A case should only be regarded as one of genuine ptosis, when the skiagram shows that the pyloric orifice has been displaced downwards (*see* fig. 175).

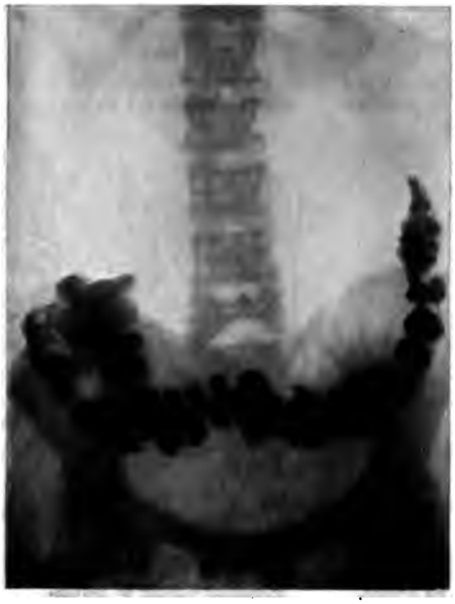


FIG. 173. — Skiagram of large intestine, twenty-four hours after taking the bismuth meal. Normal shape, slight ptosis.

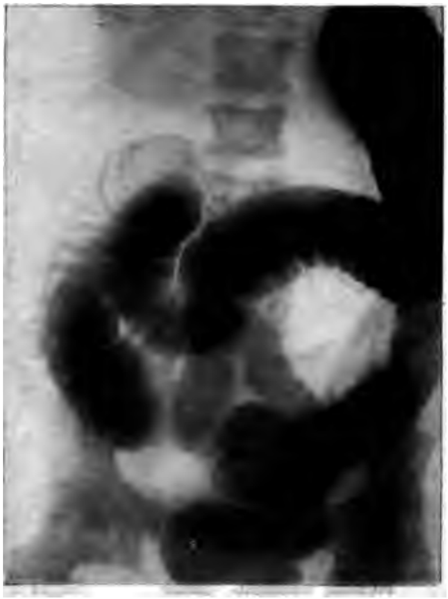
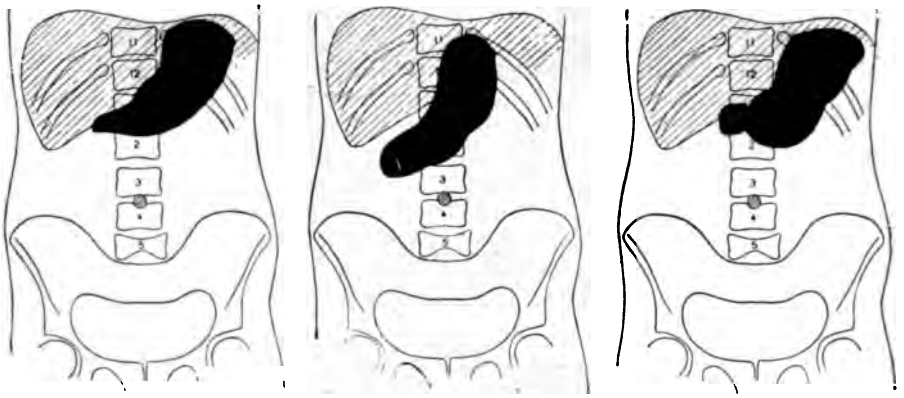


FIG. 174. — Skiagram of same case after a large bismuth injection, showing distortion in position and shape.

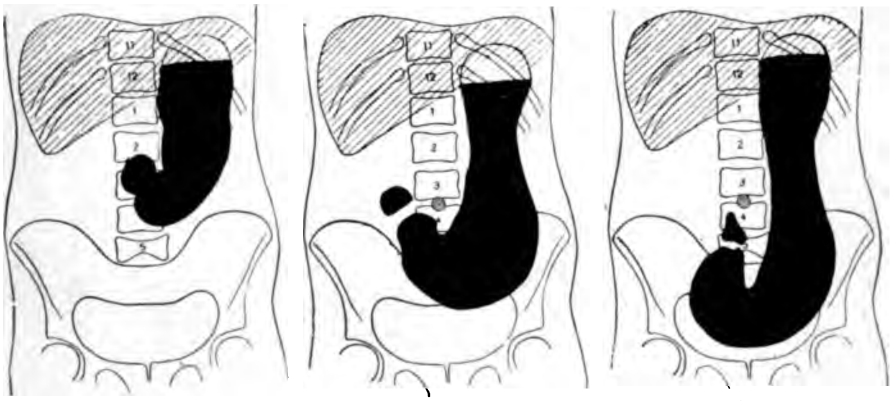
The normal position of the pylorus extends from the level of the transverse process of the first to the second lumbar vertebra. In the erect posture, it descends to the extent of one and a half vertebra, and



(*a*)
Normal stomach. Abdominal position. Bull's horn shape (infrequent).

(*b*)
Normal stomach. Usual shape. Right side position.

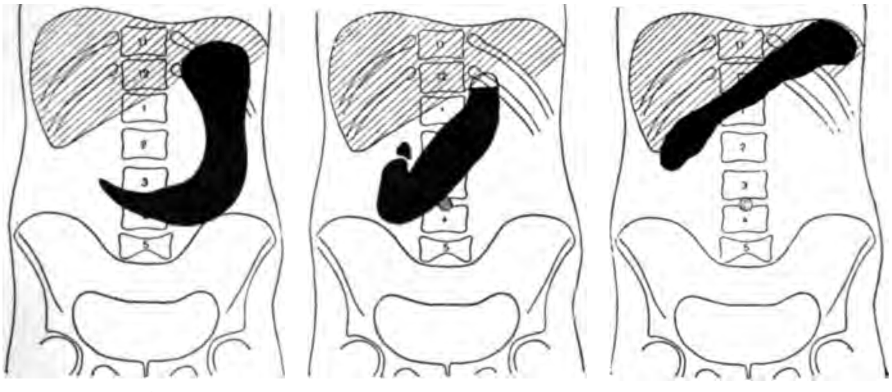
(*c*)
Normal stomach. Same case. Abdominal position.



(*d*)
Normal stomach. Same case. Standing position.

(*e*)
Slight degree of ptosis. Standing position.

(*f*)
Extreme degree of ptosis. Standing position.



(*g*)
Displacement of stomach by a cyst of pancreas.

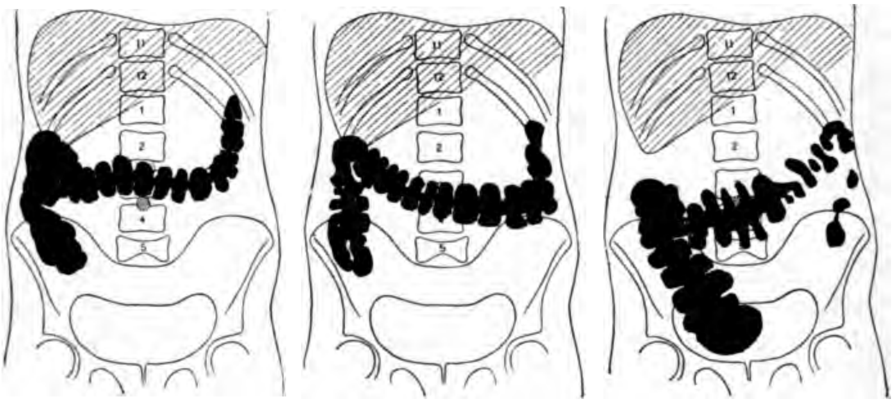
(*h*)
Displacement of stomach by a leukæmic spleen.

(*i*)
Displacement of stomach by a fibroid of uterus.

FIG. 175.—Semi-diagrammatic representation of the more important shapes and positions of the stomach.

somewhat to the middle line. In ptosis, the displacement may be double this amount.

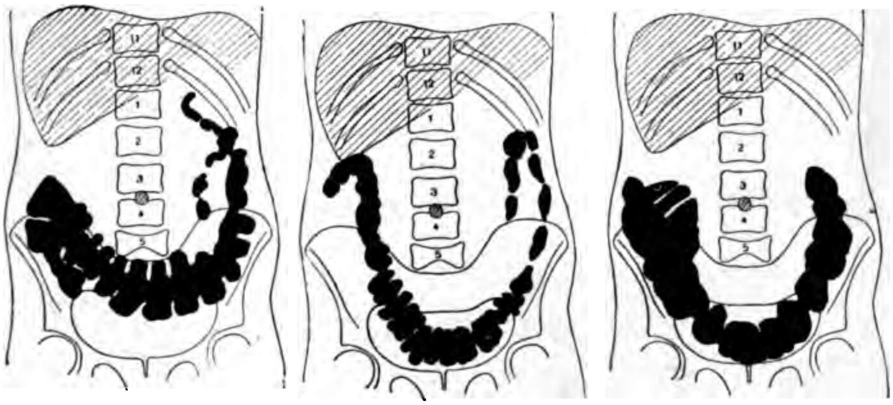
The position of the transverse colon was always looked upon as the criterion, as far as the large intestine was concerned, and this still holds good. Skiagraphy has, however, shown that the transverse colon may reach down as far as the true pelvis in consequence of its great development in length, without there being any visceral ptosis—a confirmation of a fact previously known, but to which little attention was paid. Displacement of both flexures is of greater importance; it is seen in its most marked form in the hepatic flexure, because of



(*a*)
Transverse course of transverse colon.

(*b*)
Descending course of transverse colon.

(*c*)
Ascending course of transverse colon.



(*d*)
Dependent position of transverse colon, rather low.

(*e*)
Dependent position of transverse colon, very low. Flexures at normal level.

(*f*)
Same case, with low level of hepatic flexure.

FIG. 176.—Semi-diagrammatic representation of the more important shapes of large intestine. Taken mostly twenty-four hours after administration of shadow-forming substance per os.

the greater mobility of the right kidney, The left flexure, like the left kidney, does not descend so low, even in pronounced ptosis. These conditions can only be recognised in a skiagram, with complete certainty. This shows that the position of the transverse colon varies considerably in the same patient, with the different conditions of contraction which it may present—a point which confirms results which can also be obtained from palpation.

The criteria furnished by skiagraphy in regard to gastro- and enteroptosis may be briefly summarized as follows: Low level of the greater curvature and pylorus, low level of hepatic flexure, and very frequently an encroachment of the cæcum beyond the linea innominata *down into the true pelvis, in the erect posture.*

Although these are all matters of no slight diagnostic interest it does not follow that enteroptosis affords a successful field for therapeutic activity. Nearly all the abdominal organs have been stitched up and the statement has been made that all the viscera can be restored to their normal position. This may be true anatomically, but we cannot ascribe all the numerous discomforts of "*des équilibrés du ventre,*" merely to the visceral displacement. Defective innervation of the digestive organs must bear some of the blame. This, of course, does not exclude the possibility of a vicious circle arising in certain cases, due to primary disturbance of function on the one hand, and change in form and position on the other hand—a circle which can only be broken by operative interference. But these are the exceptional cases, and the surgeon is always anxious to participate with the physician in their treatment, however anxious the patient may be for operation.

CHAPTER XXXV.

ABDOMINAL INJURIES.

FROM the surgical standpoint abdominal injuries demand careful examination, early diagnosis, and rapid decision. Lives are constantly sacrificed because diagnosis is delayed until the complete clinical picture of peritonitis is developed.

A.—INJURIES WITHOUT AN OPEN WOUND.

The cases wherein *violence has produced no open wound* present the greatest difficulties, because we rarely know the precise spot of the application of the force, and therefore the range of possible organs involved is much larger than in cases of perforating, incised, or gunshot wounds. It is therefore necessary in every case to examine all the abdominal organs, for nothing is more fatal than to overlook, for instance, a rupture of the bowel in a case wherein a renal injury is the predominant clinical feature. In examining, we must think first of the most urgent danger which is liable to follow these injuries, *i.e.*, *hæmorrhage.* If we can exclude this, we next investigate for *rupture of the gastro-intestinal tract*, and finally for an *effusion* from one of the *hollow viscera*, such as the gall bladder or urinary bladder.

(1) GASTRO-INTESTINAL CANAL.

If the injury has merely been a **contusion**, there may be no severe symptoms at first, except the initial shock. The injury may only be recognized from blood appearing in the stools, or by symptoms of slight intestinal obstruction coming on after two or three days.

Similar symptoms will be caused by injury to the portion of mesentery in contact with the bowel, or by a clean detachment of the mesentery from a coil of intestine, if circulatory disturbances short of gangrene have been produced in the intestinal wall. If the extent of mesenteric detachment has exceeded two centimetres, gangrene will, however, not usually be long in appearing.

In a case of **rupture**, the injury is not as a rule narrowly circumscribed, as with a gunshot or perforating wound. There is usually laceration of a considerable part of the circumference of the intestine, or even a whole loop may be completely torn through. The *entrance of gas into the abdominal cavity* is therefore much more likely when the injury has produced no external wound than in the case of a gunshot or perforating wound. But if the intestine was empty at the time of the injury this symptom cannot always be demonstrated.

One must never assume that there is no free gas in the abdominal cavity, as is often done, because the liver dulness is still present. The amount of free gas may be very small, and in that case it would only show itself in the highest point of the abdomen by its very tympanitic or metallic note, and by its change of situation with the altered position of the patient. These two signs are so reliable, and their method of demonstration so harmless, that we may entirely abandon puncture, as a test for the presence of gas in such cases.

The following case is instructive in this connection: A young man was caught between the buffers of two locomotives, and was brought to the hospital two and a half hours later. The liver dulness was still present. The pulse was quiet—80 per minute—temperature and general appearance normal. But there were spontaneous pains and tenderness on pressure in the epigastrium and in the left hypochondrium. The abdominal muscles were reflexly contracted. On percussion there was dulness in the lumbar regions, extending lower on the left than on the right. Liver dulness was present, but careful percussion elicited a metallic note over a very limited area in the region of the ensiform process. When the patient was turned to the right the metallic note shifted to the left; when he was turned again on to his back it returned once more beneath the ensiform process. The patient vomited once. Diagnosis: injury to intestine when comparatively empty, with exit of an insignificant amount of gas which occupied the highest portion of the abdominal cavity. Immediate operation was undertaken, which revealed a loop of the jejunum torn transversely. This was sutured and recovery followed.

This movable metallic note, which is demonstrable immediately after the injury, must not be confused with a similar change in note which occurs later on in the vicinity of the injured coil, and which is immovable. This latter is due to local meteorism, consequent upon inflammatory changes. Neither must it be confused with the collections of gas which form in the free abdominal cavity, or in encapsuled abscesses later on in the course of peritonitis. In some cases there is no exit of gas at all, as I have myself seen in a case wherein there were three complete transverse rents. This is especially true of ruptures of the jejunum; and in these cases the diagnosis must be based on the symptoms to be subsequently discussed.

The presence of *dulness* is of uncertain value in diagnosis. When the intestine is empty, a portion of the abdominal cavity is often dull, even under normal conditions, especially the left hypochondrium and the hypogastrium. Even if the dulness is to be attributed to the injury, it might just as well be due to blood as to an effusion of gastric or intestinal contents. Pronounced *defense musculaire* points to the effusion of intestinal contents, but slight muscular contraction is rather in favour of hæmorrhage. Striking pallor points to hæmorrhage, and if cyanosis begins, it is probable that the intestine has been ruptured.

If there is nothing suspicious on the first examination we should wait, watching the patient carefully, examining him again and again at short intervals. If the condition becomes at all worse it must arouse grave suspicion. If a widespread contraction of the abdominal wall persists, associated with tenderness on pressure and pain on deep inspiration, then the more certain can we be of an intestinal injury the more circumscribed were the limits of the contusion. Thus experience shows that ruptures of the intestine are especially frequent after kicks with a hoof. *It must again be emphasized that, in this early stage, one must not expect pinched features, dry tongue, distended abdomen or thread-like pulse, even if the injury has been severe. Vomiting may even be completely absent.*

We may summarize our diagnostic reflections in the following sentence: *If a patient, a few hours after an abdominal contusion, has a somewhat rapid pulse, reflex muscular contraction, tenderness on pressure, pain on deep inspiration, and manifests slight restlessness without* *simultaneous signs of a severe hæmorrhage, then the case is so suspicious of an injury to the intestine that an exploratory incision is urgently indicated, if the surroundings are suitable, as in hospital.*

The symptoms just described, apparently slight, but very expressive, may persist for twelve or even twenty-four hours without any striking changes. But then the scene changes, and vomiting, meteorism, a rapid small pulse, show us that peritonitis has set in and threatens to defy all therapeutic measures.

It should be mentioned that some patients suffer from severe meteorism after an aseptic hæmorrhage into the abdominal cavity, and this condition suggests peritonitis. Examination of the blood does not aid in the diagnosis, because there is also considerable leucocytosis in cases of peritoneal hæmorrhage. If, however, the hæmorrhage has only been moderate in amount, the pulse is quieter than in peritonitis. But in all doubtful cases, exploration is indicated.

(2) THE SPLEEN.

If an increasing area of dulness in the left half of the abdomen is accompanied by symptoms of continuous hæmorrhage, without signs of an injury to the intestine, we must think of a **ruptured spleen** although such an incident as an isolated injury is exceedingly rare when the spleen is *normal*. It is quite different when the *spleen is enlarged*, as in leukæmia, malaria, chronic congestion, and cirrhosis of the liver. In these cases the spleen is no longer protected by the ribs, and is therefore more liable to injury. The following case illustrates this.

An alcoholic female, aged 50, was found one morning dead in bed next to her husband. The autopsy showed all the signs of severe alcoholism, and revealed the cause of death in the rupture of a spleen three times the normal size, with the effusion of three litres of blood into the abdominal cavity. Numerous traces of bruises on the body and the evidence of a night scene between the deceased and her equally inebriated husband, elucidated the cause of the injury to the spleen.

The danger to which an enlarged spleen is exposed is shown by the circumstance that a malarial spleen has ruptured simply owing to palpation through the abdominal integuments.

(3) LIVER AND BILE DUCTS.

Injuries to the liver and bile-ducts are much more frequent than ruptures of the spleen, occurring even after falls from a height. The danger is caused both by the risk of *hæmorrhage* and the effusion of *bile* into the abdominal cavity. Hæmorrhage supervenes very quickly and shows itself by dulness over the right side of the abdomen, in addition to the usual signs of loss of blood. Owing to the presence of the cæcum filled with gas, this dulness is less striking than the dulness present on the left side in cases of ruptured spleen. If at the same time the liver region is sensitive to pressure, and there are radiating pains towards the right shoulder, the diagnosis is obvious. But it is not always as easy as this ; sometimes it is difficult to discover whence the blood is issuing, even after the abdomen is opened.

It is even more difficult to recognize a flow of bile into the abdominal cavity. The dulness is of much more gradual onset, and its position will vary according to the direction in which the bile is poured out. It may flow behind the stomach into the lesser omental sac, or into the lateral and inferior portions of the abdomen, or it may be limited, by rapidly formed adhesions, to the mid-abdominal region. If a diagnosis of injury to the bile ducts is based on an existing and gradually increasing fluid effusion into the abdominal cavity, without signs of acute peritonitis or of anæmia, a careful consideration of all the clinical symptoms will often indicate the approximate position of the injury. An example will save the necessity of any long disquisition.

A nine year old lad was caught under the wheel of a cart. After the subsidence of the first severe symptoms, a complete dulness of the whole upper abdominal region supervened, with subsequent bilious vomiting, partial but not complete absence of colouration from the stools, and slight jaundice. The gradual onset of epigastric dulness without anæmia pointed to an effusion of bile, and the jaundice showed that the bile was absorbed into the circulation. But as there was vomiting of bile and as the fæces retained some biliary colouring matter it was obvious that the bile duct was not torn. But the possibility of an injury to the gall-bladder or to a branch of the hepatic duct remained. The operation showed that the bile had collected in the lesser omental sac, pushing the stomach forward. This, of course, excluded an injury to the gall-bladder, so the lesion could only be a rent of a branch of the hepatic duct, with rapidly forming fibrous adhesions around the foramen of Winslow.

This observation confirms the general experience that the flow of healthy bile into the abdominal cavity is comparatively well tolerated. A fibrinous peritonitis usually develops around the effusion of bile, which shuts it off from the rest of the abdominal cavity, and if not subjected to anything but very slight disturbance, leads to spontaneous recovery. But if the effusion of bile is considerable, and if instead of reaching the intestine it becomes absorbed by the peritoneum, the patient finally succumbs to cholæmia.

It is quite different when an infected ulcerated gall-bladder bursts in the course of cholecystitis or becomes perforated by a stone. A fatal general peritonitis generally supervenes in a very short time.

(4) THE KIDNEYS.

Despite the comparatively protected situation of the *kidneys*, they are often involved in injuries which produce no external wound, especially after a fall from a height or after being run over. *Pain on pressure over the renal region*, and *blood in the urine* confirm the diagnosis, at any rate where it is possible to exclude injury to the lower urinary tract, that is to say, when there is no injury to the pelvis or perineum and micturition presents no difficulty. Sometimes the pain on pressure over the injured kidney is not very great; but on the other hand, the *reflex spasm of the lumbar muscles* on the injured side is very striking. Unilateral *renal colic* is very significant (obstruction of the ureter by a clot).

Hæmorrhage is the principal danger in renal injuries. The extent of an *extra-peritoneal injury*, at any rate, can be estimated by feeling a swelling over the renal region by means of palpation from before and behind, noting its increase and also the onset of extending dulness in the front. The most reliable conclusions are however to be drawn from the signs of an increasing anæmia, which we must not confuse with the initial signs of shock. If the swelling and dulness increase in the course of the following day, without corresponding signs of anæmia, we must assume that *urine* as well as blood is being effused, and this must be regarded as an urgent indication for interference. But sometimes anæmia may occur without any corresponding palpable change in the renal region. We must not, in such a case, assume that the blood is retained in the renal pelvis or retro-peritoneal tissue, but rather that it is flowing freely into the abdominal cavity, that is to say that the peritoneal covering of the kidney is torn through. These *intra-peritoneal injuries of the kidney* are especially prevalent among children, because their kidneys are not so closely enveloped in peri-renal fat as those of adults.

One may feel inclined to differentiate between extra and intra-peritoneal injury of the kidney on the basis of the presence or absence of the gastric and intestinal symptoms, *i.e.*, vomiting and meteorism. But caution is necessary; because gastric disturbances are sometimes noted in extra-peritoneal injuries of the kidney. This may be due to reflex causes, but it is also known that the effused blood may extend widely in the retro-peritoneal tissue and produce functional disturbance of the large intestine, with all the symptoms of obstruction. On the other hand, vomiting and meteorism, as a sign of peritoneal irritation, are not indispensable accompaniments of an intra-peritoneal rupture of the kidney. Indeed, a certain amount of blood and even of urine is well borne by the peritoneum. The formation of fibrinous adhesions, as shown by experiments on animals, acts as a defence against a persistent inflow of urine, a circumstance which justifies waiting, as long as symptoms are not on the increase.

There remain therefore two definite evidences of intra-peritoneal injury of the kidney, viz. (1) the absence of definite swelling in the renal region, with (2) the simultaneous presence of a fluid effusion free in the abdominal cavity, arising from the neighbourhood of the affected loin. But not even this absolutely assures our diagnosis, because we must be able to prove that the fluid effusion does not originate from an injury to some other abdominal organ. For instance, an extra-peritoneal contusion of the kidney may occur together with a rent in the liver or spleen. But in these cases our diagnosis cannot get beyond the range of probability.

Even if the first few critical days after an injury to the kidney have passed, the onset of local inflammatory signs and an aggravation of the general condition after an initial improvement may, at this later stage, indicate the necessity for immediate laparotomy. Tissues infiltrated by urine are especially liable to infection, and even if sepsis of the urinary tract by catheterism has been avoided, bacteria may gain access from the blood stream or from the neighbouring intestine.

(5) THE BLADDER.

Rupture of the bladder only occurs when the viscus is full, and the accident is especially liable to happen to an intoxicated person, because he tolerates an abnormally full bladder with equanimity, owing to his state of alcoholic anæsthesia, and because after a bout of wine and beer, at any rate, the bladder possesses the fulness necessary to permit of the injury.

The following is an illustrative case: An alcoholic had reached the stage when he began to assault one of his comrades. He was therefore ejected, but not in a very gentle manner. Very shortly afterwards he died, and his friends were charged with causing his death from a ruptured bladder.

The clinical picture varies according to the site of the rupture, viz., within the abdominal cavity—*intra-peritoneal rupture,* or into the peri-vesical cellular tissue—*extra-peritoneal rupture.*

Let us imagine that a patient is brought to us on account of a severe abdominal contusion. He complains of persistent strangury, but cannot micturate. Our first thought is of a urethral injury with obstruction of the passage. But no blood flows from the urethra, and the bladder is not distended above the symphysis pubis. We percuss the abdomen immediately, but elicit nothing therefrom; but while we are examining the patient, he succeeds in passing a few drops of blood-stained urine. These few symptoms suffice for the diagnosis of a recent *intraperitoneal rupture of the bladder.* We introduce an ordinary metal catheter, with rigid aseptic precautions. It enters quite easily, but only a few drops of bloody urine escape—although the patient assures us that he has not micturated for several hours. On moving the catheter about, we miss the sensation which we obtain when it is in a full bladder. The differential diagnosis lies between one of two things only—(1) reflex anuria following trauma, or (2) intra-peritoneal rupture of the bladder. The strangury and the presence of blood in the scanty urine are, however, decisive, for the latter. If the patient is seen for the first time, a few hours or a day after the injury, an additional sign is present on percussion—viz., a fluid effusion in the lower abdominal region, which, however, does not present the convex half-moon shape at the superior border, which is found in a free intra-peritoneal effusion. The catheter enters the empty contracted bladder, but after moving it about therein, we may suddenly find that it becomes quite free, and a large amount of fluid escapes, which on chemical examination, is shown to be highly albuminous. This means that the catheter has gone through the rent in the bladder into the abdominal cavity, and has drawn off the collection of urine, mixed up with albuminous exudation. The dulness which was present immediately before the catheter was passed has now diminished. If the patient is seen in a still later stage, there will be found some slight peritoneal irritability with increasing effusion of fluid in the abdominal cavity. If attempts have been made to pass a catheter there is every prospect that this peritoneal irritability will rapidly become a definite peritonitis. We should, however, avoid this result by sewing up the bladder before any large collection of urine takes place within the peritoneum, or before uræmic or septic symptoms arise.

Extra-peritoneal rupture is quite different. The patient complains principally of strangury, but he passes much more urine than in cases of intra-peritoneal rupture, and the catheter shows that the bladder is not completely empty. The fact that there is an injury to the bladder is indicated by the presence of strangury, while the urethral canal is quite permeable, and by the presence of blood in the urine without symptoms pointing to the kidneys or urethra. It is just in this circumstance that careful attention will demonstrate a symptom concerning which Dittrich stated : "*If air enters the bladder by means of the catheter we shall detect a limited area with a metallic note immediately over the symphysis.*"

The symptoms hitherto mentioned are apparently mild, but they are rapidly complicated by the onset of infiltration of urine into the pelvic cellular tissue which causes dulness over the symphysis. This may be followed by infiltration and phlegmonous swelling of the lower abdominal region, and even by signs of uræmia. Signs of peritoneal irritation may supervene, but these play quite a subordinate part.

Though typical cases of both forms of injury are easily distinguished, the diagnosis may be very difficult *in ruptures of slight extent*, e.g., when in a case of fractured pelvis, a fragment of bone penetrates the bladder. In all doubtful cases the wisest plan is to explore the bladder.

B.—INJURIES TO THE ABDOMEN WITH OPEN WOUNDS.

Open injuries are generally caused by stabs, incisions, or by bullets. Diagnosis is facilitated because the position and direction of the injury to the soft parts give some definite information about the possibility or probability of injury to one or other abdominal organ. The following case shows that one must take into consideration the course of origin of the injury in order to decide upon its direction.

A young man was hit in the gluteal region by a Flobert bullet, which came from a weapon carelessly placed on the floor behind him. He complained at once of abdominal pain, which his friends attributed to something he had eaten—a quite probable cause, as the accident happened on a public holiday. It was not thought at first that a bullet in the gluteal region could cause abdominal pain. Thirty hours later he was brought to the hospital with symptoms of acute peritonitis. The wound of entry was found above the left gluteal fold, but no wound of exit could be seen. The bullet had struck posteriorly and inferiorly, and must have gone through the left great sciatic foramen to reach the abdominal cavity. As a matter of fact the X-rays revealed the bullet in the right lower abdominal region, and the operation showed that there was a double perforation in the last coil of the small intestine.

(1) GUNSHOT WOUNDS.

Gunshot wounds of the stomach and intestine caused by the ordinary small firearms differ from subcutaneous lacerations by the smallness of their extent. In the case of bullets of very fine calibre, the opening is so small that it is very difficult to discover it even at the operation. This also applies to the small-calibred bullets of modern military firearms, which cause little deformity, except those which strike diagonally, or are fired at close range. But on the other hand, several loops of intestine are often shot through at the same time.

The symptoms of gastric or intestinal injuries are naturally the same as those caused by violence without an external wound. But the symptoms may be so very slight when the diameter of the intestinal wound is small, and the bowel is empty, that spontaneous healing is much more likely to take place than in the case of subcutaneous lacerations. Advantage is taken in war of this favourable outlook, where the impossibility of opening the abdomen at the right moment compels us to dispense with this procedure. It is quite different in civil practice, when as a rule, timely surgical assistance is available for the injured. We must not, therefore, wait and see what the man's luck is going to be, but must give him the best opportunity of recovering by means of an immediate laparotomy. *Every abdominal gunshot wound which by its direction may involve the stomach or intestine is so suspicious of having caused injury to the intestinal canal if the projectile cannot be found in the abdominal integuments that it is absolutely urgent to bring the patient to the hospital at once.*

We have just said "if the projectile cannot be found in the abdominal integuments." This is, however, not an instruction to undertake a search for it, on the spot. Formerly the surgeon used to take a probe out of his pocket-case, insert into the wound, and if it reached any depth would exclaim "The case is hopeless." Of course it is quite clear that the probe can often inform us whether the injury has perforated, but it is equally clear that the procedure may arouse a fresh hæmorrhage, tear through protective adhesions, and infect a wound which has hitherto remained aseptic. It would be a safer plan to lay the gunshot track freely open, after thoroughly cleansing it, in order to see whether the bullet still remains therein or has gone through the peritoneum. But this procedure is only permissible when everything is in readiness to undertake a regular laparotomy and suture the stomach or intestine. Otherwise, we should leave our probe in the pocket-case, with the other instruments, apply a first aid dressing and send the patient to hospital forthwith. I have never seen projectiles discharged at close quarters lodge in the abdominal integuments. This could only occur with a defective charge, or when the bullet has been stopped by pieces of clothing. To enlarge the wound of entry in a hurry simply affords opportunity for infection before laparotomy can be undertaken, and deprives the surgeon of the important indication which the direction of the track gives him in his search for the injured intestine. We should limit our activity to a careful estimate of the state of affairs—the distance and direction of the bullet—and leave the wound alone, despite the urgent demands of the patient and his friends for the "instant removal of the bullet."

Gunshot wounds of the liver, kidneys and bladder are diagnosed on the same principles as injuries of these organs without an external wound.

(2) STABS AND INCISED WOUNDS.

Stabs and incised wounds which penetrate the abdominal wall are so suspicious of intestinal injury that it is our duty to open the abdomen, as in the case of gunshot wounds. How do we ascertain whether such an injury has perforated the abdominal wall? The rules laid down in regard to gunshot wounds apply equally to *punctured wounds*, i.e., a "search" may only be instituted if it can be immediately followed by a laparotomy. In all other circumstances the indications are—first aid dressing, careful history and removal to hospital.

If there be no protrusion of bowel in *incised wounds*, we should, when circumstances permit, separate the wound with clean hands, and this will often show whether it has penetrated to the abdominal cavity. In cases of extensive incised wounds, and of the notorious laceration inflicted by the horns of bulls, the intestines are frequently protruding when the patient is brought for treatment. In such circumstances no attempt must be made to replace the bowel; a large first aid dressing must be provided, and the injured at once sent into hospital, after having been given opium as an intestinal sedative.

It is not always easy to discover the perforated coil, even at the operation. For example: A fat man was stabbed in the left lumbar region with a slaughtering knife. No symptoms of internal hæmorrhage, no signs of intestinal injury, and no blood in the urine. The position of the wound accorded with an injury to kidney or spleen; the length of the knife was sufficient to have wounded the bowel. On opening up the wound, an injury to the lower pole of the kidney was discovered, and laparotomy revealed the expected complete rent of two-thirds of the circumference of the descending colon at the renal flexure, although this is a rare event.

Stabs of the large intestine are, as a rule, much rarer than knife wounds of the small intestine.

CHAPTER XXXVI.

ACUTE INFLAMMATION WITHIN THE ABDOMINAL CAVITY.

BEFORE attempting to discover the origin of any inflammatory disease within the abdomen, we first make sure that an inflammatory process really exists. This is, however, not always easy.

Let us consider two typical cases. A patient suddenly begins to complain of abdominal pains and to vomit. The temperature is slightly raised, the pulse is accelerated, the breathing is shallow, and almost exclusively thoracic in type; the abdomen is not distended—it is, however, tender on light percussion, and responds thereto by muscular contraction. Pressure over the lumbar regions is painful either on one or on both sides. Neither flatus nor stools pass; but there is no visible peristalsis. The patient complains of a constant dull pain, which varies in severity, but does not cease completely. There is no doubt at all that *peritonitis* is beginning in this case. Let us compare this with the other case. The illness also begins with abdominal pains and vomiting; but the pulse is quiet and full, except at the actual time of nausea and vomiting; the temperature is normal, the breathing is not accelerated, nor particularly shallow. The abdomen is not distended, or only slightly so; during the periods of repose it is not tender either to pressure or percussion, and the abdominal muscles do not markedly contract on palpation. Neither flatus nor stools pass. An attack of pain occurs from time to time, during which peristalsis can be seen if the abdominal wall is thin. The attack hardly lasts a minute; when it is over, the patient feels well until the next attack warns one that some severe disturbance is in progress. Evidently this is a case of *intestinal obstruction.* The differential diagnosis between peritonitis and intestinal obstruction is usually quite easy in the early stage. But this early stage must be most carefully watched, and if we are called too late for this we must obtain its history as accurately as possible, because as the illness advances the differentiation becomes more difficult. In peritonitis the abdomen distends gradually, the temperature is not always raised, but often becomes sub-normal, and symptoms of functional or mechanical obstruction are frequently added to those of simple inflammation. In obstruction, on the other hand, the pulse becomes small and rapid, as the case progresses, the temperature may rise, and the abdomen remains tensely tympanitic even in the intervals which are free from pain. The pain eventually becomes persistent, and the mechanical obstruction is supplemented by intestinal paralysis, or even by peritonitis.

If the initial symptoms lead us to the conclusion that some inflammatory irritation of the peritoneum exists, we must not be content with the vague diagnosis of "peritonitis," but must endeavour to trace its sources as quickly as possible. Careful investigation and observation at the beginning are of the greatest importance for this purpose, for once the peritonitis has become generalized we are no more able to discover its origin than we can detect the origin of a fire when the whole house is ablaze. In this stage it is generally too late to successfully overcome the inflammation.

The method by which inflammation of a limited area spreads over the whole abdomen follows various types:—

(1) In the first group, we are confronted within the first few hours by a diffuse, and generally a simple toxic irritation of the peritoneum, presenting all the symptoms of a mild generalized peritonitis; but there may be no specially sensitive spot to indicate the origin of the mischief. In fact, in these cases, the patient is unable to say where

the pain started. On opening the abdomen a serous, or even a somewhat turbid exudation containing leucocytes, but always odourless, is found; but no organisms can be cultivated therefrom. In a few hours, or at any rate after a few days' interval, the general symptoms abate, the spontaneous pain as well as the pain on pressure limit themselves more and more to an area which corresponds to the original seat of the mischief. Indeed an abscess is forming, and cure may take place either by its absorption or by perforation into the bowel (fig. 178, *a—h*).

(2) In the second group the initial symptoms resemble the above but the reaction of the peritoneum is more acute. The exudation soon contains bacteria—usually from the second day in cases of appendicitis—and often has an offensive odour if the infection has come from the intestine. Subsequently the peritonitis diminishes in some places, in others fibrinous investments form, whereas at the periphery of the abdomen there is a tendency to the development of encapsuled abscesses, entirely independent of the original area of the disease (fig. 178, *k*), the so-called "residual abscesses" (Restabszessen). We agree with Sprengel, &c., in interpreting in this sense, the "progressive fibrino-purulent peritonitis" of Mikulicz and Burkhardt.

(3) In other cases, after the general peritonitis subsides and the inflammation has become localized, a fresh and more severe attack of peritonitis may supervene, which signifies that either the primary or a residual abscess has burst into the peritoneal cavity.

(4) In the severest cases the symptoms are generalized from the beginning, and remain so until death. The sero-purulent peritonitis merges into the diffuse purulent form, and the clinical symptoms depend more upon the virulence of the micro-organisms than upon the anatomical conditions.

Some general remarks are required to introduce the discussion of the individual forms of peritonitis.

The *previous history* is often very valuable. Sufferers from appendix trouble and gall stones, who have already experienced attacks, recognize the seat of their malady. In females, a reliable history is indispensable, but is often difficult to obtain. For instance, in criminal abortion, as well as in spontaneous abortion which the patient is reluctant to confess, she often endeavours to make her friends and medical attendant adopt the view that the case is appendicitis.

If some general disease has preceded the peritonitis we should think of typhoid perforation. These perforations are more frequently overlooked than one imagines, because the symptoms are partially masked by the underlying disease. A few years ago a well-known surgeon died from an undiagnosed perforation occurring in the course of an undiagnosed typhoid fever, although he was surrounded by physicians and surgeons—which ought to be very consoling to the general practitioner.

The *age* and *sex* of the patient must be taken into consideration.

In the *male sex*, up to the age of 20, the vermiform appendix is so frequently at fault, that this ought to be our first thought, in every case of peritonitis, even if it has not started on the right side. After the age of 20 such comparatively rare causes as perforated gastric or duodenal ulcers may suggest themselves, or exceptionally, perforation of a tubercular ulcer of the small intestine. From the age of 40 or 50, or even earlier, we should also think of the gall-bladder. Indeed, with the advance of age, the probability of the gall-bladder being responsible, and not the appendix, increases. Intestinal perforation due to cancer falls within the same age period. I have twice seen cases of calculous cholecystitis imitate appendicitis before the age of 20, and these cases were both in girls aged 18.

In the *female sex*, in early age, we have to think, in addition to perityphilitis, of pneumococcal peritonitis—a rarity among boys. This often develops quite independently of the vermiform appendix, and we shall deal with it again later on. After puberty, once the hymen is ruptured, all the inflammatory processes which may start in the sexual organs must be taken into consideration. An intact hymen, as McRae rightly says, points to appendicitis in doubtful cases.

We begin our *physical examination* with a *general view of the entire patient*.

Nothing is so reassuring as a face in repose. Nothing fills us with so much apprehension as the restlessness which the patient manifests about everything; the haste with which he persuades us that nothing ails him, that he feels well, although his pulse is hardly perceptible. Flushes on the face and ears indicate that he is within the grasp of peritonitis, and cyanosis, first to be detected on the finger nails, signifies that the toxæmia is well advanced. Jaundice, sometimes nothing more than a slight pigmentation of the sclerotics, is not rare in general peritonitis, and it is of bad omen. A moist tongue is a good sign, although it may be coated. A dry tongue, even uncoated, shows that mischief still persists somewhere, that the system has not yet mastered the infection. We conclude from rapid shallow breathing in which the nostrils participate that the peritonitis is on the increase, or when the face is generally flushed that some complication exists in the lung. Quiet, painless breathing shows that the inflammatory area has become limited. A full pulse, slow in relation to the temperature, is a good sign; a small soft pulse, rapid in relation to the temperature, is of bad import; a pulse of normal amplitude, but soft and rather dicrotic, indicates an inflammatory focus in the abdomen, which has not yet been overcome.

The age and sex of the patient must be taken into consideration while estimating the pulse-rate. Sometimes a peritonitis may be fully developed, especially in men, and the pulse does not exceed 90. On the other hand, in children, a pulse-rate of 120 to 140 is not of serious significance. If the pulse persists at 130 and over, in adults, the condition is very grave.

The temperature as taken in the axilla is of but little value. Often enough, it does not exceed the normal, even in fatal peritonitis. But it is important, when compared with the rectal temperature; for the greater the divergence, the worse the outlook.

We now proceed to the *examination of the abdomen*. We must first empty the bladder with a Nelaton catheter : even if the patient has micturated in our presence. Patients with abscesses in the lower abdomen do not completely empty the bladder, and it often happens that the full bladder is mistaken for an effusion "because the patient has only just micturated."

We seize the opportunity of using the catheter, to examine the urine, and if albumen or much indican be present, we conclude that the illness is severe. Bile pigment indicates disease of the bile ducts, and sugar suggests some pancreatic affection.

In estimating meteorism, we must remember that the abdominal circumference in young people and in the male sex is, *ceteris paribus*, smaller than in females. In percussing, very light movements are required in order to detect a superficial thin layer of effusion.

Direct, one finger percussion, known as *palpation-percussion*, is a valuable method, in patients who are not too stout. The whole abdomen is percussed lightly with the middle finger applied flat, by which device, dulness can be *felt* as well as it can be *heard*. It is best always to employ both methods of percussion and compare the results.

The presence of a metallic note over individual loops of intestine is of special significance. It is an important sign of a strangulation, a kink, or of local peritonitis; the same applies to circumscribed crepitations always heard over the same place, or to the peristaltic metallic note so carefully described by Wilms and Leuenberger. Palpation must be very cautious. It is better to remain uncertain of the precise extent of an abscess, rather than to break down protective adhesions through want of care. The more carefully one proceeds the more reliable will be the result of the examination. If the percussion is carefully conducted reflex muscular contraction will cause much less interference and liability to error, than when a rough method is adopted. We cannot entirely avoid this muscular contraction; its presence is very well known, but is not sufficiently appreciated as an aid to diagnosis.

This contraction, or "defense musculaire" which we have already mentioned in connection with abdominal injuries, occurs in the muscles which cover over the diseased area and it constitutes the first sign of inflammatory irritation of the parietal serous membrane. It diminishes with the course of the disease, or limits itself to those areas which the inflammatory process has involved, in its spread. In cases wherein the whole abdominal cavity is inundated by infective material, particularly after extensive gastric perforation, the entire abdominal wall becomes firmly contracted, or at least contracts as soon as the slightest touch impinges upon it. In perityphlitis, the contraction is usually limited to the right lower abdominal region, and if the process spreads towards the loin then the lumbar muscles of the right side contract. If the lumbar muscles of the left side also contract on pressure, we may be sure that the peritoneal irritation has spread to that quarter. This is the more pronounced, the more the parietal peritoneum is involved in the inflammation, and it may therefore be entirely absent when the inflamed area is deeply situated, e.g., in meso-cæliac or pelvic appendicitis. The respiratory fixation of the muscles in the vicinity of the diseased area is also due to this reflex contraction, as shown by Küster. It is noteworthy that this reflex contraction is much slighter in puerperal cases than in those infected from the intestine.

In examining for sensitiveness to pressure, we must not be misled by expressions of pain which nervous people will manifest, just like spoilt children, even when pressure is made on healthy organs. If the peritoneum is really inflamed, pain will be elicited when the hand is suddenly removed after gentle pressure.

It is necesary to make a brief observation here, regarding the perception of pain in the abdominal organs. Although Harvey and Haller had already shown that the viscera do not respond to ordinary irritation, by pain, nevertheless the site of morbid pain sensations at any rate, was localized in the viscera themselves, until a few years ago. Lennander opposed this view, as a result of his careful investigations, and he sought to explain all sensations of pain by dragging or friction of the nerves of the anterior or posterior abdominal wall, including the mesenteric attachments. Ross, and subsequently Head, assumed that local pain sensibility may occur in the abdominal viscera themselves, giving rise, reflexly, to sensations of pain in a corresponding area of the abdominal wall. Head went a step further and endeavoured to show by numerous investigations, that disease of a thoracic or abdominal organ produced severe localized hyperæsthesia in a very definite segmentary area of skin integument, corresponding to the abdominal organ. If we adopt this theory, we must not any longer attribute local hyperæsthesia to pure functional disturbance (hysteria), without considering the possibility of some deep-seated disease. James Mackenzie has made numerous observations which have led him to certain conclusions of no small diagnostic interest, and which we will here briefly summarize. Like Lennander, he assumes that stimuli such as pinching, pricking, and burning are inadequate to excite the sensation of pain in the abdominal organs themselves. Even an adequate stimulus like contraction does not suffice to excite a sensation of pain, under normal circumstances. But if these stimuli exceed a certain degree they irritate, in the sense of a viscero-sensory reflex, sensory fibres in the spinal cord, which come from the corresponding area of the abdominal integument, and they also irritate, as a viscero-motor reflex, motor fibres which proceed to corresponding muscle segments. Thus, if we pinch an intestinal segment, we excite no pain. But if the intestine contracts in an unusually marked degree, the irritation reaches the spinal cord and stimulates therein spinal pain fibres, causing the brain to appreciate pain in the corresponding spinal segment. This pain is referred to the corresponding area of abdominal skin and not to the abdominal viscus, from which the irritation arises (referred pain). The muscles of the corresponding segment may be simultaneously irritated, causing reflex abdominal contraction.

Although this view is to some extent hypothetical, it explains Lennander's observations, which are in themselves very accurate, much better than his own theory.

Finally we should make a rectal or a vaginal examination, which will clear up the condition of the female genital organs, afford information as to the existence of a pelvic abscess, and allow us to decide whether it should be opened from above or below. The more deeply it is situated the more evident it will be, not only by the bulging but also by the œdematous swelling of the mucous membrane—feeling like velvet—and by the profuse discharge of jelly-like mucus. Tenesmus is rarely absent.

But as previously observed, our attention must not be devoted exclusively to the abdomen. There is often a recurrence of fever in the course of peritonitis, after some improvement has begun. The tongue becomes dry again. Nothing is, however, to be felt in the abdomen ; but the remarkably rapid breathing, the diffuse congestion of the face, and the dicrotic pulse will forthwith suggest some lung complication to the experienced observer. Careful examination will reveal either a **pneumonia** or a **pleurisy,** or both together, usually as a metastatatic process, but in the case of pleurisy it may be the result of direct extension through the diaphragm.

Sometimes cerebral symptoms appear in the course of peritonitis. These may be due to a metastatic process like abscess, or to sinus thrombosis, but rarely to meningitis. Occasionally these symptoms are the result of inanition.

Hitherto our discussion has been limited to methods of diagnosis which are always available for the general practitioner ; but attempts have been made for a number of years to base indications for diagnosis and prognosis on the condition of the white corpuscles in cases of inflammatory disease—especially of the abdominal cavity.

As far as the *number* of white cells is concerned, it is established that a considerable increase, apart from digestion, generally indicates an inflammatory process. But a similar increase may occur after an aseptic hæmorrhage into the abdomen, so that leucocytosis is of small value as an aid in differential diagnosis. On the other hand, leucocytosis is absent in certain infective diseases (typhoid), and also in the most severe forms of peritoneal infection.

More definite indications are yielded by a *differential count of the white cells* and from special changes in the neutrophil polynuclear leucocytes. Schindler takes into consideration the number of myelocytes; Arnet counts the nuclei, or fragments of nuclei, of the neutrophil polynuclear leucocytes, and draws conclusions from their greater or lesser fragmentation. Sondern finds an aid to prognosis in the proportion of the leucocytosis in general, to the percentage of the polymorpho-neutrophil leucocytes. All these various methods give some information concerning the defensive activities of the organism. But it is indispensable, in the case of them all, that the examinations should be made at regular intervals, just like the taking of temperature. This, however, requires adequate laboratory equipment and a certain amount of technical experience, in addition to more time than a practitioner can usually devote to individual patients, and therefore these methods must be practically limited to hospitals and clinics. The practitioner does not possess the necessary leucocyte curve at the moment he is called to the patient to enable him to arrive at a decision, and to wait for the preparation of one may frequently delay surgical intervention until it is too late. As Kocher once said, the main thing in appendicitis is not to demonstrate a hyper-leucocytosis, but to prevent its onset by early operation. On the other hand, a leucocyte curve is of value for the hospital surgeon in deciding, for instance, whether a late case of appendicitis requires operation, or whether a second operation is necessary in the course of a suppurative disease of the abdominal cavity.

In arriving at a decision in cases of peritonitis, we must bear in mind a fact which is often forgotten in practice, that all cases do not present a typical, clinical picture. Sometimes on operating we find the intestines swimming in pus, although the clinical symptoms may not have led us to anticipate any severe disease. The pulse remains good, the reflex muscular contraction is slight. There is neither vomiting nor intestinal paralysis, and the patient recovers despite the gloomy outlook. There are two reasons for these exceptional cases. In the first place the peritonitis is not so generalized as it appears to be. The convolutions of the small intestine, although bathed in pus, are not infected between the individual loops, being protected by the omentum and by fibrous adhesions (Lennander's peripheral peritonitis). The second reason is just as important, if not more so: the slight virulence of the pus organisms. For instance, the pneumococcus is comparatively harmless in children, and a central peritonitis due to it may run an excellent course although the condition found at operation leads us to think that the case must prove fatal. Other micro-organisms may also be equally innocuous, in exceptional instances. We need only think of cases of acute peritoneal sepsis which are fatal before anatomical changes have had time to form in the serous membrane, to realize that the prognosis of peritonitis depends more upon the virulence of the micro-organisms than upon the extent and intensity of the anatomical changes.

We now proceed to construct a diagnosis from the conditions found upon physical examination. The most frequent of these conditions may be classified as follows:—

(*a*) Symptoms of pain without perceptible changes.

(*b*) Symptoms of pain, with signs of general peritoneal irritation without definite localization.

(*c*) Peritoneal irritation, with circumscribed changes.

A.—ABDOMINAL PAIN WITHOUT PERCEPTIBLE CHANGES.

The first group of symptoms, characterized by spontaneous pain and by local pain on pressure, without any obvious change, is responsible for most of the errors of diagnosis. We may hesitate between hysteria, crises of tabes, a mild attack of appendicitis, an attack of mucous colic, renal or biliary colic, some disease of the female genitalia, acute intestinal obstruction, and even an inflammatory disease of the thoracic organs.

Suspicion of some **hysterical condition** will be aroused by any striking contrast between the complaints made and the actual condition found. Formerly, hyperæsthesia of the skin suggested hysteria, but as we know now that disease of deeply situated organs may be recognized by superficial hyperæsthesia, we must be cautious in the interpretation of this sign. Simultaneous deep and superficial hyperalgesia *may* be due to hysteria, but is *not necessarily* so. But "appendicitis" is *certainly* hysterical if the hyperæsthesia is exclusively confined to the skin.

Elevations of temperature which do not correspond with the pulse and general aspect of the patient, should excite suspicion. Some hysterical individuals are able to manipulate the thermometer for their own purposes, until they convince a surgeon that it is really necessary to remove their appendix. This deception can be eliminated by taking the temperature per rectum.

We must examine for **tabes** in every case, wherein the other conditions are reconcilable with this diagnosis.

A female, aged 50, became ill suddenly one night with severe abdominal pain, which suggested an acute gastric or intestinal perforation. Although physical examination proved negative, the question of laparotomy was contemplated, but the history made one hesitate. Her husband had died twenty years ago from paralysis, she had had one miscarriage, and a swelling on the skull which had been cured by potassium iodide. Further examination showed that the pupil reflex was lost, and that the knee-jerks were absent—the only signs of tabes. The abdomen was not opened. Rectal crises occurred later on, in confirmation of the diagnosis.

In **renal colic** either a normal or enlarged kidney, sensitive to pressure, is felt, or, at any rate, pronounced reflex contraction of the lumbar muscles is present. The pain radiates into the genital organs and even to the thigh, and the corresponding testicle may be abnormally sensitive to pressure. If the case is not seen until the cessation of the pain, some information may be obtained by examining the urine.

Gall-bladder colic is distinguished from renal colic and appendicular pain by the situation of the sensitive area, which is internal to the outer border of the rectus, and at the level of, or above the umbilicus. It may easily be mistaken for duodenal pain; but gall-stones usually occur in females and duodenal ulcers in males.

Temporary strangulation of a **hernia**, which has been overlooked both by patient and doctor, may be mistaken for a mild attack of appendicitis, quite apart from appendicitis in a hernial sac.

On one occasion I removed the appendix of a middle-aged woman on account of the history of appendicitis, and the diagnosis made by the medical attendant during the attacks. But the attacks returned, and I eventually discovered the cause in a right-sided crural hernia, which could hardly be detected by the most careful examination.

Attacks of **mucous colitis** or **entero-spasm** are often mistaken for inflammatory diseases. Muco-membranous colitis, with its ileo-cæcal pain, tenderness on pressure over the appendix region, and its transitory pulse of collapse and vomiting may completely resemble an attack of appendicitis.

We merely refer to the matter here, but will discuss it more fully in connection with *chronic appendicitis*. Other symptoms of pain in the large intestine, the significance of which is often difficult to decide, will be dealt with together later on.

The gums should always be examined in unexpected colic, lest a case of *lead colic* be missed.

We have already referred to **intestinal obstruction**, and will discuss it in detail later on.

The attacks of pain induced by adhesions, by anomalies of position and of form, belong to this group. If the history and skiagram do not afford any definite guidance, a positive diagnosis can only be obtained if the transitory kink, twist or band leads to genuine intestinal obstruction with a subsequent operation, or examination *post-mortem*.

One should not forget the possibility of **painful menstruation** in females.

The somewhat rare conditions of *intra-abdominal menstruation*, and intermenstrual ovarian hæmorrhages ought to be thought of, if they could be diagnosed. Such cases have been operated on, in mistake for appendicitis. Our experience suggests that these conditions should be thought of, in the case of a virgin suffering from symptoms like appendicitis with a leucocytosis of 20,000 or over, and only a moderate rise of temperature. If the hymen has been ruptured, the diagnosis is more likely to be extra-uterine pregnancy.

Finally it must not be forgotten that little children usually refer the pain of **pneumonia** or **pleurisy** to the abdomen. In adults also, diaphragmatic pleurisy is manifested by pain on one side of the abdomen and muscular contraction.

The pain of **tubercular caries** rarely extends so far into the abdomen as to cause serious diagnostic difficulties.

B.—DIFFUSE PERITONITIS WITHOUT LOCALIZATION.

The *second group* of cases confronts us with different questions altogether. The physical condition—accelerated pulse, pyrexia, general tenderness, a rigidly contracted musculature or a distended abdomen, an effusion, either free or badly localized—shows that the peritoneum is acutely inflamed. The diagnosis of nervous disease or colic is no longer possible.

But it is just here that there is the danger of confusing **obstruction** with peritonitis. We have already stated the main points of the differential diagnosis at the beginning of this chapter. The effusion, which so often accompanies strangulation by bands, adds to the resemblance, especially as peristalsis is frequently inhibited in strangulation, thus banishing the chief symptom of difference between peritonitis and obstruction.

We may similarly be deceived by strangulation due to a pre-existing, but frequently overlooked **tubercular peritonitis.** The tubercle produces the exudation, and the strangulation evokes the acute symptoms.

A young girl, aged 18, hitherto quite well, except for some vague indigestion, was suddenly attacked by abdominal pain, vomiting and fever. Two days later I found her in the following condition : high fever, rapid hardly perceptible pulse, accelerated respiration, dry tongue, red flushes on face, distended sensitive abdomen with dulness in the dependent part. Everything pointed to a generalized peritonitis subsequent to appendicitis. But there was one sign which caused hesitation. When I arrived the patient was sitting half up, and she resumed this posture without any difficulty during the examination. A patient with severe peritonitis never does this; and therefore some other diagnosis had to be found. This was suggested by the fact that there were two tubercular uncles, and that the patient had previously suffered from digestive trouble. The inference was that the case was one of an overlooked tubercular peritonitis on which an acute obstruction had supervened—the latter condition having rapidly reached the stage of intestinal paralysis, diffuse distension, and rapid small pulse. Hence this picture of peritonitis. The diagnosis was confirmed at the operation.

We may also be deceived by a **"meso-cœliac" appendicular abscess** lying between or behind the coils of the small intestine (fig. 178*d*). These abscesses are situated too high to allow of their being felt per rectum. If the forwardly displaced loops of small intestine are so distended that the abscess cannot be felt through the abdominal wall, one usually thinks of generalized peritonitis, or of obstruction, unless the consideration of the whole course of the illness leads to a correct diagnosis.

There are yet two rarer conditions which may lead us astray, viz., **acute pancreatitis with acute fat necrosis** or pancreatic hæmorrhage, and **blocking** of the **mesenteric vessels.** The former condition is suggested when the symptoms are mainly confined to the upper abdominal region, and the patient is a stout man of middle age or older. The presence of sugar in the urine is a valuable aid to diagnosis, but the patients usually succumb before the onset of glycosuria.

The blocking of the mesenteric vessels may concern an artery, or a vein, even the whole portal vein. In the case of arterial blocking, the cause is either embolism, or thrombosis ; when a vein is blocked the cause is always thrombosis. The anatomical result is infarction, or gangrene of a more or less considerable section of the intestine. From the outset the clinical signs sometimes partake of the character of peritonitis, with persistent deep, dull pain and effusion, sometimes that of obstruction. Blood in the stools, and the vomiting of lumps of blood, and the retention of flatus, should arouse suspicion. The existence of valvular disease, indicates embolism, especially if the patient has manifested other signs of embolism. Diseased blood-vessels, and general defects of the circulation would point to thrombosis. As a rule, the embolism is first recognized, either at the operation or at the autopsy.

Having established the diagnosis of acute infective peritonitis, we must endeavour to determine its origin. The considerations with which we introduced the discussion of peritonitis will be of great assistance, as also the remarks to be made in the next section on localized inflammatory processes. When in doubt, we must suspect appendicitis, at any rate, in civilized countries. In other regions it may be otherwise, because this disease is not equally prevalent everywhere.

It is stated that only whites suffer from appendicitis in China and South Africa. It is uncertain whether this is a matter of race or of nutrition.

C.—LOCALIZED PERITONITIS.

If there are clear symptoms, pointing to a localized area of inflammation, our task is much easier, whether general symptoms are present or not. We will proceed topographically, starting with the upper portion of the abdominal cavity.

(1) EPIGASTRIUM.

The most frequent origin of inflammation in the epigastrium is in the **stomach or duodenum,** and the cause is either a gradual or sudden perforation of an ulcer, or more rarely of cancer. **Pancreatic hæmorrhage and pancreatitis,** which we have already referred to, should also be mentioned. Exceptionally, a hepatic abscess may be situated in the middle of the epigastrium.

(2) RIGHT HYPOCHONDRIUM.

In temperate climates the **bile ducts** are mainly responsible for inflammation in this region, with extension into the liver substance in the form of septic cholangitis. In the Tropics, **abscess of the liver** is the most frequent occurrence. This does not exclude the possibility of hepatic abscess in temperate climes, but then the abscess is generally the result of gall-stone disease, which is predominant clinically, or it is metastatic in nature, and recedes into the background in comparison with the other signs of the pyæmia. Rupture of a duodenal ulcer must next be mentioned. If the rupture has been gradual it causes a localized inflammation, if it has been sudden it causes generalized peritonitis.

We shall see later on that, occasionally, a vermiform appendix may wander into this region.

(3) LEFT HYPOCHONDRIUM.

Inflammatory processes occurring in this vicinity, are nearly always the result of **perforated gastric ulcers. Abscess of the spleen** is very rare, but it has been observed especially in tyhoid fever. In cases of appendicitis, secondary abscesses often occur in the left hypochondrium, or travel up into this region from below.

(4) LUMBAR REGIONS.

Retro-peritoneal inflammation in the lumbar region is mainly concerned with the **kidney.** If the inflammation has extended so far forward that we must assume that the *peritoneum is involved*, then on the right side, we should first think of **appendicitis** (fig. 178 *g*). This diagnosis is obvious if the ileo-cæcal region is also involved. On the other hand I have seen experienced practitioners in doubt, even where this area has been quite free, and when the whole process has been confined to the lumbar region. The lumbar variety of appendicitis is, however, not always, intra-peritoneal. When the appendix is in this position, it is usually either entirely or partially retro-peritoneal, so that the peritonitic symptoms are not predominant, and the disease process tends toward the back, appearing as a phlegmon of the cellular tissue attended by high fever (fig. 178, *l*).

Very significant of this form of phlegmon, which usually contains gas-forming anaerobic bacteria, is the change of the reddened skin into a yellowish or brownish hue. It cannot be a mere accident that I have seen so many of these cases end in pyæmia.

Appendicitis is often the cause of apparently primary abscesses of the *left* lumbar region. In these cases, the appendix lies in a very dependent position, and the primary abscess, which is not palpable from above, originates in the true pelvis (fig. 178, *e*), and then tracks towards the left.

It should finally be mentioned that **parametritis** often extends towards the lumbar region.

(5) LOWER ABDOMINAL REGION.

(*a*) If an inflammatory process occurs in a female, *exactly in the middle line*, our first thought must turn to the **uterus** and its vicinity, and we must not be misled either by the married or single state of the patient. If we do not at once ascertain that the disease has followed a confinement or miscarriage, we enquire about the last menstruation, and the more hesitating the reply the stronger are our suspicions. Experience shows that nearly all infections which follow miscarriages are due to the use of the sound in procuring abortion. Sometimes it happens that an apparently inflammatory swelling situated directly in the middle line of the lower abdominal region is really due to an *extra-uterine gestation*, concerning which more will be said later on. If the clinical history and vaginal examination exclude disease of the genital organs, we must look elsewhere for the cause of the trouble, and again we fall back upon the **appendix,** which is often responsible for a primary abscess in the middle line.

In rare instances a **perforated Meckel's Diverticulum** causes a median abscess, or one situated rather more to the right beneath the umbilicus. This is also the favourite place for the pointing of an abscess due to **pneumococcal peritonitis** in little girls. If these abscesses are not opened in time, they eventually burst through the navel (fig. 177). They are distinguished from other abdominal abscesses by their remarkable softness. Abscesses due to perforation of Graser's diverticulum of the sigmoid, are occasionally situated centrally.

Ovarian cysts with twisted pedicles or which have **suppurated** are usually situated in the middle line. A superficial examination is liable to mistake them for abdominal abscesses, or for appendicitis if they are situated towards the right side. Their sharply defined upper border should usually establish the diagnosis. Twisting of the pedicle is comparatively frequent, while suppuration of an ovarian cyst is rare. The former condition is very sudden in its onset, and to some extent is distinguished from a suppurating ovarian tumour by the course of the temperature.

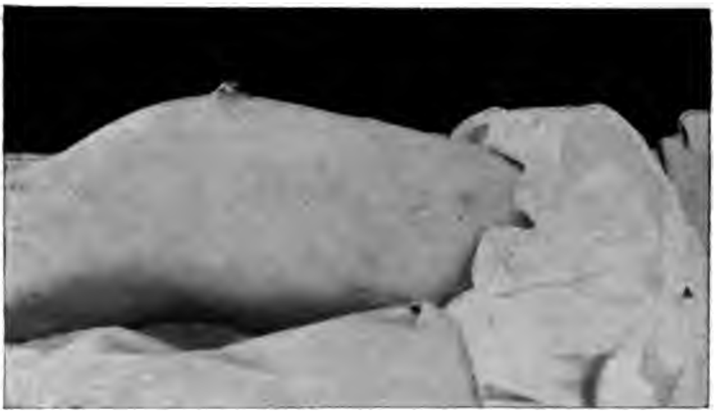


FIG. 177.—Pneumccoccal peritonitis on the point of bursting through the umbilicus.

Abscesses in the *abdominal wall*, **below the umbilicus** and above the **symphysis**, must not be mistaken for abdominal abscesses. The sub-umbilical abscesses usually arise from infective disease of the peritoneum, while those above the symphysis originate either in some infective trouble of the urinary tract or from osteomyelitis of the pubis. The sensation that the abscess is immediately beneath the hand, and the absence of intestinal symptoms with the occasional presence of bladder disturbance, makes the diagnosis quite easy.

(*b*) If the resistance is *at the side* we are fully justified in thinking of the appendix on the right, and we shall often have to assume the same origin, even if the abscess is on the left side, in males. In females we must think, in addition, of salpingitis, parametritis, extra-uterine pregnancy, and more often than in males, also of the gall-bladder.

It should be added that the position of the diseased area, in appendicitis, is not always at McBurney's spot, between the anterior superior spine of the ileum and the umbilicus; indeed it is usually not there. The appendix, as we shall see later on, varies very much in its position (fig. 178, *a*, *d*, *g*, *h*).

We first consider the *history*. If abdominal pain and vomiting constituted the onset, and similar attacks have preceded, the case is probably **appendicitis** or may be cholelithiasis. If the symptoms have come on with the menstruation, or if they have apparently brought it on early, this should not tempt us from the first diagnosis. But if the illness has followed a miscarriage or a confinement and is not accompanied by signs of peritonitis, we can only assume that it is a case of phlegmon of the broad ligament (fig. 178, *p*), *i.e.*, *pelvic cellulitis* or *parametritis.*

The following case serves as an example for diagnosis: A woman, aged 38, was brought in, for a suspected strangulated hernia.. She was three months pregnant, and had a threatened abortion a few days before. No hæmorrhage now. There was a hard swelling in the right hypogastrium, reaching below Poupart's ligament. There were prominent lung symptoms and blood-stained sputum. Many examinations were made and many diagnoses suggested. Contradictory statements regarding origin of symptoms. Suspicion of criminal abortion, with injury to right vaginal vault and phlegmon of right cellular tissue, venous thrombosis and pulmonary embolism. Uterus apparently not affected, as there was neither pain nor hæmorrhage. *Per vaginam*, uterus three months pregnant, somewhat fixed on the right. Circumscribed, hard infiltration of mucous membrane in right vaginal vault. Right-sided parametritis. Speculum showed that the portio was uninjured, that there was a thrombosed varix with two wounds of the mucous membrane, a few millimetres each, in the right vaginal vault. These could have been inflicted by a knitting needle or similar instrument. The threatened abortion was therefore a hæmorrhage from a perforated varix, upon which followed infective thrombosis, phlegmon of pelvis and thigh, pulmonary embolism and multiple abscesses,—and finally the patient recovered.

A cylindrical or irregularly circular sharply-defined swelling at the border of the true pelvis and the iliac fossa, which has taken months or years to form, is a **salpingitis.** In its development, frequent, but not severe inflammatory attacks occur, and careful investigation will generally reveal a history of gonorrhœa or tubercle. The disease may be unilateral or bilateral (fig. 178, *o*). The history of gonorrhœa can only be obtained by confidential questioning of the husband, if there be one. If we are informed that irregular hæmorrhage occurred after one or two periods had been missed, and that the lateral swelling which we detect has been accompanied by severe pain, collapse and perhaps by vomiting, we shall seldom go wrong in diagnosing a **ruptured tubal gestation,** or a tubal abortion (fig. 178 *q*).

We derive most information from *physical examination*. By this method we are able to define morbid conditions above, towards the

FIG. 178.

Diagrammatic Representation of the more Important Phases and Forms of Appendicitis, and of some Considerations in Differential Diagnosis.

Green = abscess contiguous to the anterior abdominal wall.

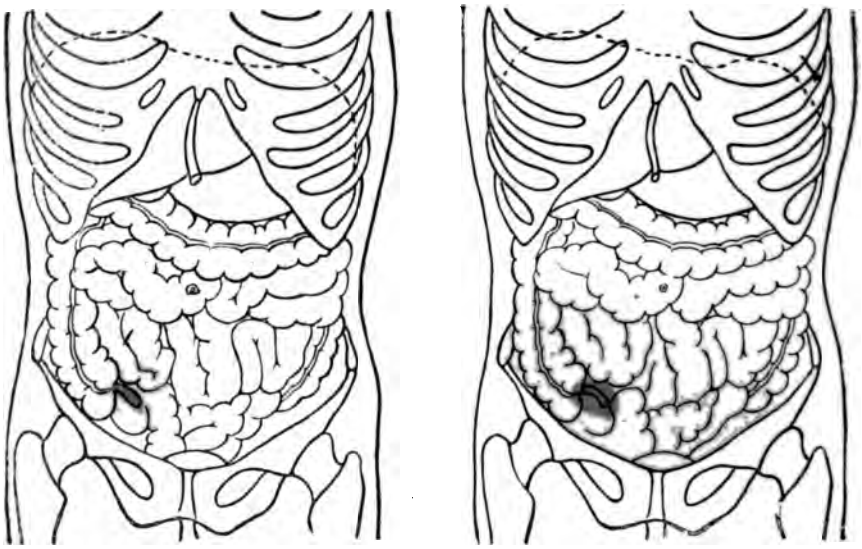
Shaded green = abscess covered by intestine.

Yellow = serous or sero-purulent exudation, aseptic on first day, infected early exudation on second day.

Yellowish-green = sero-purulent exudation, full of bacteria.

Blue = extra peritoneal abscess.

Red = blood effusion.

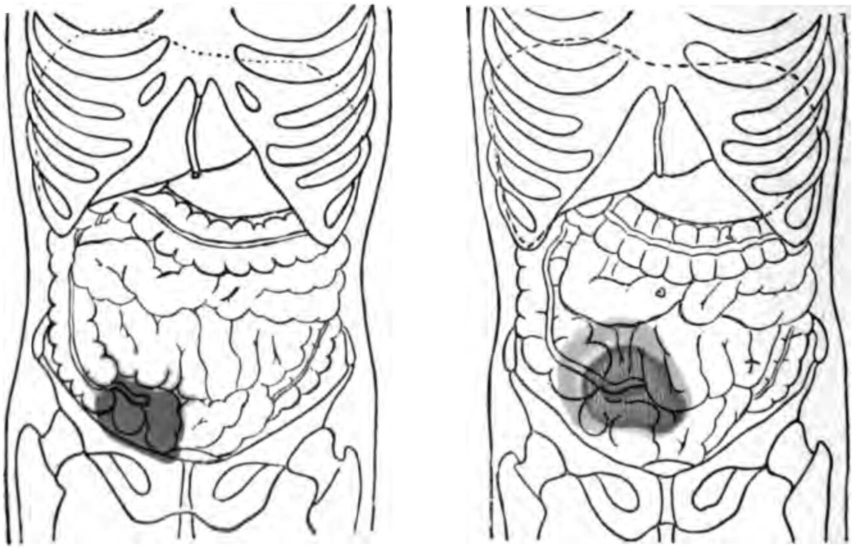


(*a*)

Appendix forwards and inwards. Appendicitis, antero-parietal. First stage. Purulent appendicitis. Localized serous peri-appendicitis. (Early exudation beginning.)

(*b*)

Same form. Second stage, second to third day. Purulent peri-appendicitis beginning. Extensive early exudation.



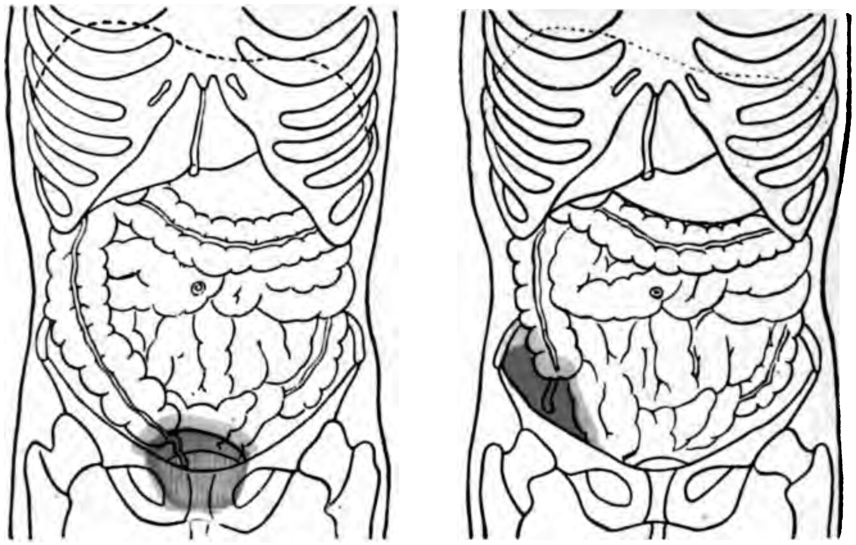
(*c*)

Same form at end of first week. Third stage. Large antero-parietal encapsuled abscess. Early exudation has subsided.

(*d*)

Same stage, but appendix situated in convolution of small intestine. Abscess covered by intestinal loop. Meso-cœliac appendicitis.

FIG. 178.

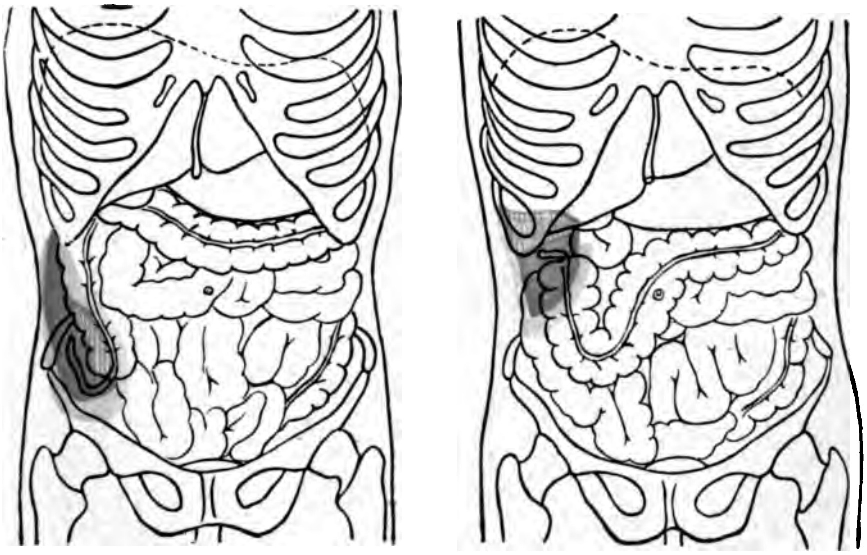


(*e*)

Same stage. Appendix in true pelvis. Pelvic appendicitis.

(*f*)

Same stage. Appendix situated outwards and downwards. Ileo-inguinal appendicitis



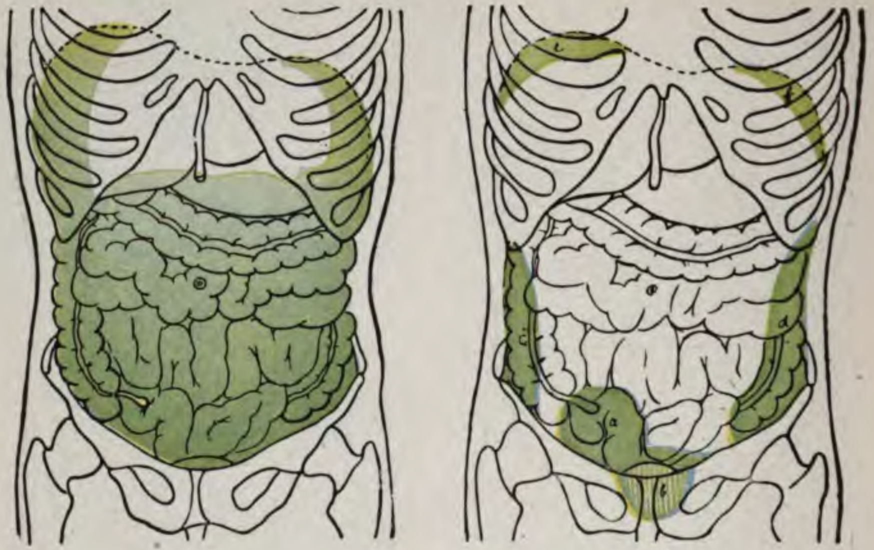
(*g*)

Same stage. Appendix directed upwards and outwards. Abscess partially behind cæcum. Ileo-lumbar intra-peritoneal appendicitis.

(*h*)

Same stage. Cæcum and appendix drawn upwards. (Beginning of left-sided transposition of colon. Common ileo-cæcal mesentery.) Sub-hepatic appendicitis. (Abscess in same position when cæcum is normal, but when very long appendix is drawn upwards and outwards.

FIG. 178.

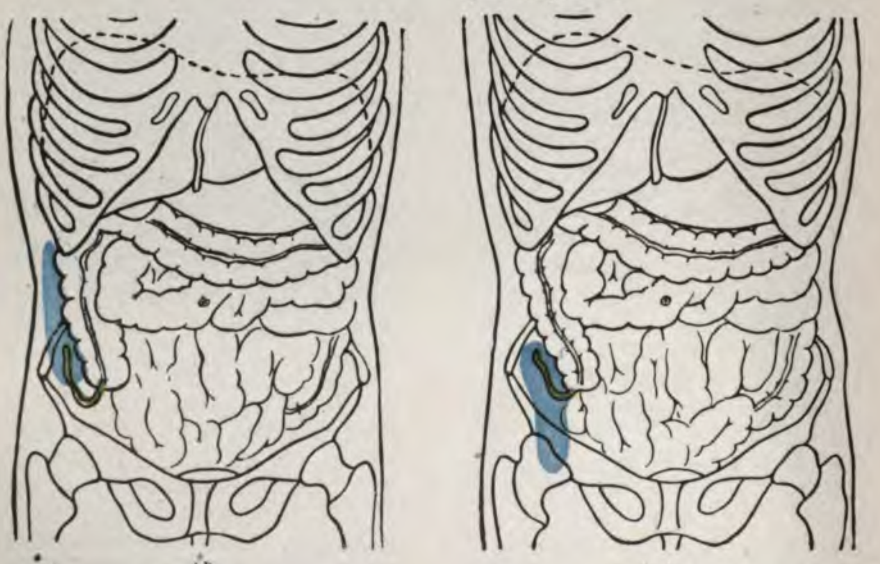


(*i*)

Diffuse sero-purulent peritonitis, in a very virulent infection, large perforation or gangrene of appendix, or secondary rupture of encapsuled abscess. This form of peritonitis is fatal, or recovery only occurs with abscesses remaining.

(*k*)

Multiple abscesses (residual) after general sero-purulent or purulent peritonitis. The figure shows the most common positions. These abscesses frequently intercommunicate, thus *a* and *b*, often *a* and *c*, or *a* and *d*, &c. The "progressive fibrinous purulent peritonitis" of Mickulicz and Burckhardt depends upon this process.



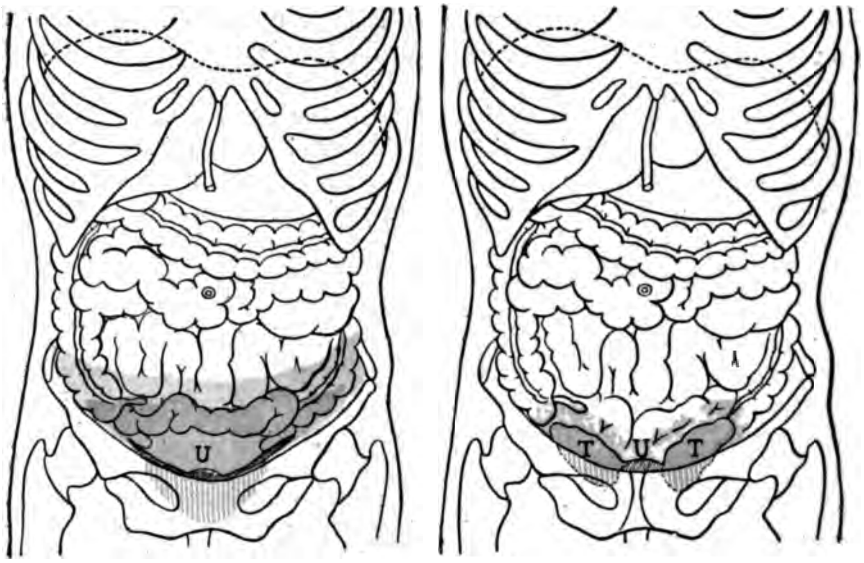
(*l*)

Appendix inclined upwards and outwards. The abscess is extra-peritoneal, sub-serous and finally became superficial in the form of a phlegmon in the lumbar region. Lumbar appendicitis or ileo-lumbar sub-serous appendicitis.

(*m*)

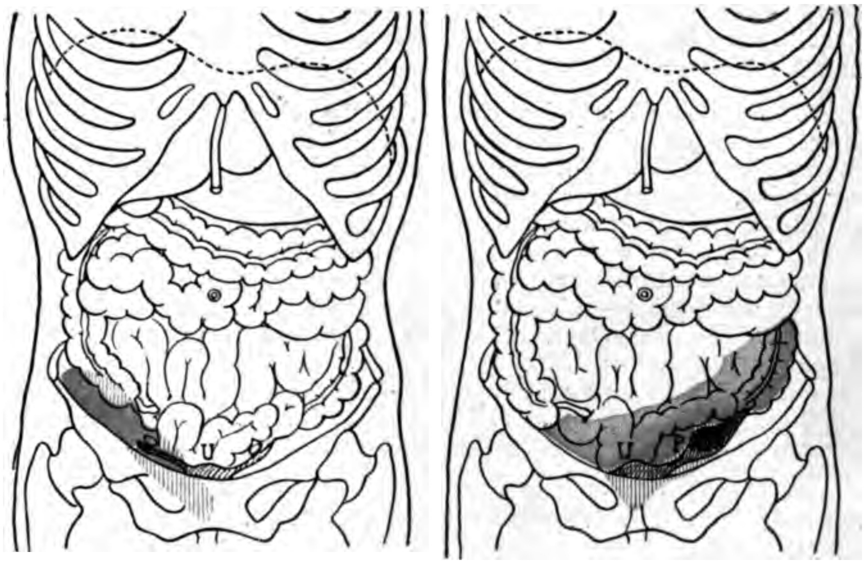
The abscess is under the iliacus fascia. Ileo-inguinial sub-fascial appendicitis. The abscess may reach the thigh by tracking under Poupart's ligament.

FIG. 178.



(*n*)
Purulent peritonitis, exciting serous peritonitis in the vicinity. U = uterus.

(*o*)
Bilateral pyosalpinx, with some serous perisalpingitis.



(*p*)
Parametritis extending as far as Poupart's ligament.

(*q*)
Rupture of a pregnant tube. P = placenta, with a peri-tubal hæmatoma around.

gall bladder, as well as those below, towards the pelvic organs. If the resistance is more clearly limited below than above, and if the maximum point of pain on pressure is high up we should think of **disease of the bile ducts.** If the gall bladder is greatly enlarged and inflamed, this point of maximum pain may be displaced below the line joining the anterior superior spine to the umbilicus, especially if the adjacent loops of intestine are involved in the inflammation. Jaundice naturally points to biliary disease, but not unconditionally, any more than the absence of jaundice is an argument against it. The age of the patient, whether male or female, is of significance, as the probability of cholelithiasis increases with age.

If the morbid process tends downwards, we decide upon **appendicitis** if the pain and the maximum point of tenderness on pressure are found above the true pelvis, while the symptoms are definitely less, lower down. On the other hand, we diagnose **parametritis,** if the swelling and tenderness have started close to the uterus, whence they have spread upwards towards Poupart's ligament, or even to the pelvic fossa or lumbar region. Vaginal or rectal examination may detect a perityphlitic exudation reaching into the true pelvis, *behind* the uterus, or an extra-peritoneal parametritic exudation *at the side* of the uterus, displacing it towards the healthy side, and more or less fixed to the pelvis. In a case of extensive **pyosalpinx,** a more or less sausage-shaped, sharply defined resistance can be felt from above, and its connection with the uterus can be made out by bi-manual examination. But even apart from this, the sharply defined limitation of the structure facilitates its distinction from an acute perityphilitic abscess, unless a fresh exacerbation of peri-salpingitis be present.

The following is a typical case. A girl aged 20 was seized at night, after a festive occasion, with "appendicitis," and was admitted within twenty-four hours with the symptoms of a commencing diffuse peritonitis. Temperature was 104·8° F., face was remarkably flushed. Indefinite resistance was felt on the right side, *per rectum.* As the flushed face was very unusual for appendicitis, and as the height of the temperature was still more unusual for the first day, we thought of rupture—sub coitu ?—of a right tube, and the operation confirmed the diagnosis. The pus contained a pure culture of *Staphylococcus aureus.*

Cases wherein an acute appendicitis becomes engrafted on an old salpingitis are not very rare, but it is quite impossible to unravel the maze of symptoms and make a diagnosis.

There may not be any less difficulty in differentiating between appendicitis and a **ruptured tubal gestation.** The swelling is situated midway between the position of an appendicular abscess and a parametritis. It is intra-peritoneal like the former, but connected with the uterus like the latter. It often reaches as far as the appendix, so that the roof of the blood cavity is formed by the appendix, cæcum and lowest loops of the small intestine, all combined.

On one occasion I found a shrivelled fœtus of about ten weeks gestation tucked away with a thrombosed placenta under the cæcum.

It will of course be understood that the oval swelling close to the uterus is only *one* of the symptoms of ruptured tubal gestation, or of tubal abortion, *i.e.*, the *peri-tubal hæmatoma*. If the history is indefinite or unreliable, it may be very difficult to distinguish it from a unilateral pyosalpinx in which peri-salpingitis has occurred—as gynæcologists know very well. In these circumstances there may be a striking absence of anæmia.

If the hæmorrhage is severe or is repeated, then the hæmatoma is no longer lateral, but becomes *retro-uterine*, and should rarely be confused with an appendix abscess in the pelvis. But if there should be any doubt, extra-uterine gestation is confirmed by softening of the vaginal cervix, enlargement of the breasts, the exit of drops of milky fluid when they are squeezed, anæmia and slight jaundice. The presence of fever is no contra-indication. A glance at the patient's ears is the most rapid indication of the degree of anæmia. Their marked pallor is as significant of a severe internal hæmorrhage as their blueness is of peritonitis, other symptoms being equal. If some abnormal swelling can be felt in Douglas' pouch in the first twenty-four hours, the case is probably a hæmatocele, because appendicitis does not lead to an abscess in Douglas' pouch so soon. If, on the other hand, the history points to extra-uterine pregnancy, and the tumour is strikingly movable, we must think of an unruptured tubal gestation or of an abdominal pregnancy. Too energetic an examination may cause a severe hæmorrhage in such a case.

The condition of the *abdominal muscles* is important in a doubtful case. The degree of their reflex spasm depends upon the nature and intensity of the irritation, aud the extent to which it has involved the anterior abdominal wall. It is most pronounced in appendicitis, less so, or absent in salpingitis and ruptured tubal gestation. In the latter case, it depends upon the extent of contact of the hæmatoma with the abdominal wall, and the sensitiveness of the individual towards an intra-peritoneal effusion of blood. It should also be remarked that individual differences in reflex irritability, and the special characteristics of the micro-organisms concerned, play an important rôle, even in suppurative diseases. The blood examination is of value, in so far as a great decrease in the hæmoglobin and red corpuscles indicates hæmorrhage. Curiously enough a high degree of leucocytosis also indicates hæmorrhage. Our experience has been that the leucocytosis of aseptic intra-peritoneal hæmorrhages is more intense than that of inflammatory processes. A leucocyte count of 20,000 to 25,000, in cases wherein the signs of infection are but slight, may be regarded as an indication of a fresh intra-abdominal hæmorrhage.

If the displacement of gas from the descending colon towards the cæcum, by pressure upwards from the left of the pelvic fossa, causes pain in the appendix, this always means appendicitis, according to Rovsnig, or, at any rate, some inflammatory process involving the cæcum. I agree with Hausmann, that—at any rate in many cases—the pressure is communicated to the inflamed area, not by the column of gas in the large intestine, but through the distended small intestine.

Diseases of the left side of the pelvis require mention, although they only half belong to the surgeon. He shares the diseases of the female genitalia with the gynæcologist, on account of the frequency of errors of diagnosis. The physician occasionally refers to him cases of **sigmoiditis,** *i.e.*, localized subacute, or acute, inflammation of the sigmoid flexure. This condition should be thought of, when a sausage-shaped swelling is felt in the left lower abdominal region, in association with symptoms of colitis: diarrhœa, mucus discharge and blood. It is still a medical disease in this stage, but if signs of peri-sigmoiditis, *i.e.*, irritation of the peritoneum, supervene, the cases become surgical and the question of operative assistance arises. We shall refer to this again in another connection.

(6) TRUE PELVIS.

To discuss inflammations within the true pelvis is apparently an intrusion into the province of gynæcology. We may, however, disarm this criticism by beginning with the *male sex*. It often happens that nothing can be felt in the whole abdomen, and then it is found that Douglas' pouch is occupied by an inflammatory swelling. What may this be? It is most probably an **appendicular abscess,** originating in an appendix hanging down into the true pelvis. But I have found there an **intussusception of the small intestine,** which I looked upon as an appendicitis until the operation. An inflamed coil of small intestine, obstructed by a **gall-stone** is often found in the true pelvis. **Cancer of the rectum** and Graser's diverticula may give rise to peri-rectal suppuration. Finally, there occur abscesses arising from the urinary tract, which are mainly to be attributed to **prostatic suppuration,** or to **suppurating diverticula,** in prostatic patients.

With the exception of prostatic suppuration, all the above conditions also apply to the *female sex*, who also suffer from the special diseases associated with the female genitalia. Some controversy has raged around the proper limitations of the work of the surgeon and gynæcologist. In order to avoid this, I will only mention here the association of **pregnancy or the puerperal state with appendicitis.** When pains occur in the hypogastrium of a pregnant woman we naturally connect them with the pregnancy, but the possibility of appendicitis must not be overlooked. This combination is most unfortunate, as it generally leads to abortion, or miscarriage. If the pregnant uterus forms a portion of the abscess wall, as is usually the case, rupture of the abscess is almost inevitable, and therewith fatal peritonitis also. The midwife or doctor is usually blamed for this, unless an autopsy throws light on the tragedy. I have seen a similar catastrophe at the end of a normal pregnancy.

For this reason, one should not shirk removing, during pregnancy, an appendix threatened with inflammation. If an abscess has formed it is imperative to open it before abortion or labour occurs.

It is sometimes very difficult to distinguish exacerbations of acute (generally of gonorrhœal origin) salpingitis, and localized thrombosis of the pampiniform plexus, which occasionally occur in the puerperal period, from attacks of appendicitis, especially when there is a previous history of appendix seizures.

CHAPTER XXXVII.

SUB-PHRENIC ABSCESS.

If a patient has continuous high fever without any obvious suppuration in any of the easily accessible regions of the body, we should bear in mind the various possibilities of sub-phrenic abscess. The existence o such an abscess is highly probable, if the patient has recently had any inflammatory disease within the abdominal cavity. About half the cases of sub-phrenic abscess originate in appendicitis; the rest are due, in the abundant experience of Körte, to affections of the stomach, liver, spleen, kidneys, pleura, ribs, intestine and pancreas —in descending proportion.

The main aid to the diagnosis of sub-phrenic abscess is to bear it in mind and search for it. We shall describe a typical case before detailing the symptoms.

A young man passed through a severe attack of perityphlitis, which was operated on, in the stage of widespread peripheral peritonitis. All the symptoms gradually abated. The temperature rose again two months after the beginning of the illness, but examination with the view of detecting a sub-phrenic abscess revealed nothing. But as the fever persisted, the patient was re-examined. The lower border of the liver was at first in the normal position, its dulness did not extend upwards above the usual limit; the condition of the lung was normal and there was a complete absence of subjective symptoms A small patch of broncho-pneumonia on the left side misled us and prevented us from making an immediate exploratory puncture on the right side. Eventually the liver dulness began to extend upwards and its lower border downwards, and slight pain on breathing supervened. This confirmed our suspicion. Screen examination by X-rays showed normal movements of the right arch of the diaphragm, but its shape was remarkably semi-spherical and it appeared to be very high. Exploratory puncture and immediate operation demonstrated a sharply localized sub-phrenic abscess, which caused considerable upward bulging of the diaphragm.

The diagnosis is rendered very difficult by the fact that percussion over the lung as well as over the abscess may yield very varying results. The lung may give a normal note, or a dull note if there be any associated pleurisy, whereas a sub-phrenic abscess will be tympanitic if it contain gas; otherwise it will be dull. The situation of the abscess is not constant, sometimes it is confined to the upper surface of the liver, sometimes its position is more forwards, at others it is situated backwards and downwards, in which case it resembles a peri-renal abscess. We will treat this subject in two parts, corresponding to the fundamental difference of the presence or absence of pleural effusion.

(1) SUB-PHRENIC ABSCESS WITHOUT PLEURAL EFFUSION.

If the abscess contains little or no gas, as in the previously described case, the only local sign is that the edge of the liver is lower, whereas its upper border of dulness is higher. The lower limit of the healthy respiratory sounds is also pushed upwards, corresponding to the liver dulness. There may be more or less definite signs of compression, but they may be quite absent. How can we distinguish this physical condition from pleural effusion? The degree of the downward displacement of the liver and the bulging of the lower part of the thorax, in sub-phrenic abscess, are too variable to be of any value as differentiating signs. Very marked signs of pulmonary compression and infiltration point to empyema; if these signs are absent they are in favour of sub-phrenic abscess. But it is more important to realize that the upper border of the dulness in sub-phrenic abscess is convex, whereas in empyema it is rather horizontal or runs obliquely towards the spine. A screen examination by X-rays is of great value, and its result furnishes even stronger evidence. The shadow cast in empyema is either horizontal, or oblique towards one side or the other, mostly towards the spine, whereas in sub-phrenic abscess it is convex upwards. But the history is most conclusive. Empyema usually follows some disease of the lung; a sub-phrenic abscess is the consequence of an infective process within the abdomen. But as a secondary pleurisy is of frequent occurrence in this latter condition, we must take into consideration the manner in which the secondary illness has started. A secondary pleurisy arises after the manner of an embolism, and sets in with more or less sudden and severe respiratory difficulty; but a sub-phrenic abscess develops slowly, and does not as a rule cause pain until it has attained a definite size. The pain is also much duller than the pleuritic pain which makes the breathing so difficult. If a pleurisy should subsequently develop into a sub-phrenic abscess, it will be obvious from the chronological order of the symptoms that the latter was not the original trouble.

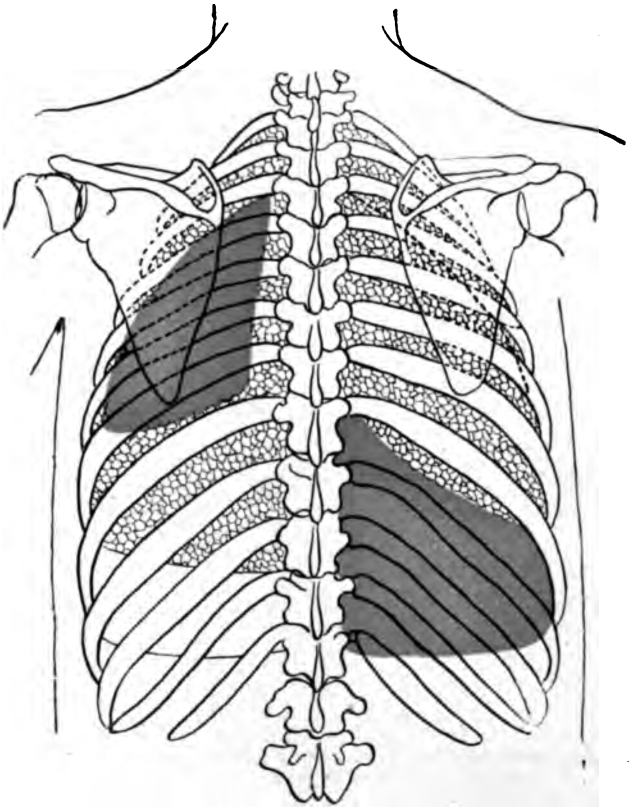


FIG. 179.—Green = pus in contact with the chest wall. Collections of pus in contact with the chest wall. On the right side, the usual basal emypema; on the left, interlobar.

In any case we should not be satisfied with the demonstration and evacuation of a serous pleural effusion, if the symptoms have set in gradually and a high temperature persists. If the effusion is sterile, the presence of a sub-phrenic abscess is at any rate extremely probable.

The diagnosis is easier if the abscess contains much gas, for percussion will yield the well-known zones : below, dulness corresponding to the liver and the fluid contents of the abscess, then a tympanitic note due to the gas, and finally the normal note of the lung (fig. 180). This also applies to the left side, *mutatis mutandis*. An abscess which contains no gas is more easily recognizable on the left than on the right, because of the extensive dulness and the displacement of the spleen downwards; sometimes also by the upward displacement of the heart. If the abscess contains gas the three zones just mentioned can be detected.

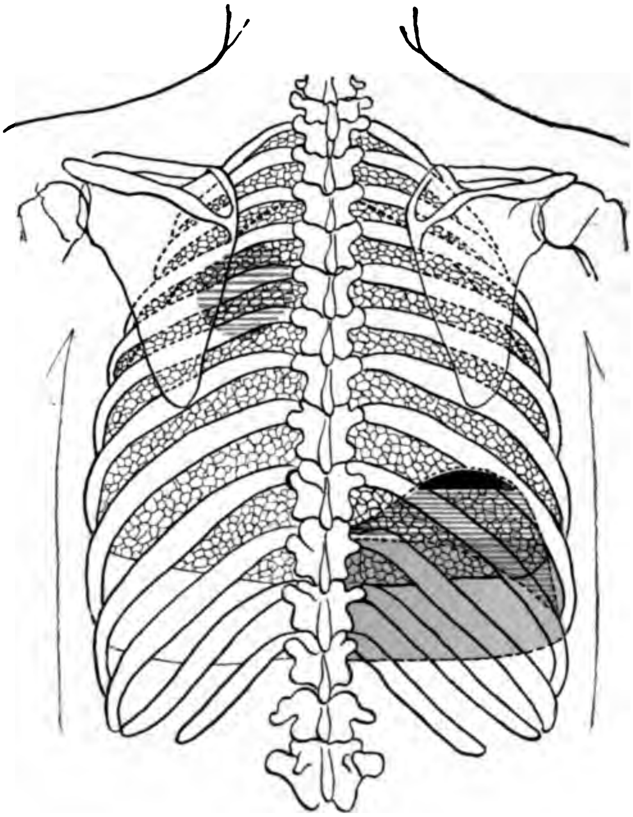


FIG. 180.—Shaded green = pus beneath lung and diaphragm. Yellow = serous effusion. Black = air vesicle. The chest wall not in contact with collection of pus. On the right, a sub-phrenic abscess with gas vesicle and serous pleural effusion; on the left, an abscess of lung.

The large gas-containing abscesses described of old are seldom seen nowadays, because they are not allowed sufficient time to develop.

(2) SUB-PHRENIC ABSCESS WITH PLEURAL EFFUSION

The conditions are much more complicated if there is a pleural effusion in addition to the sub-phrenic abscess. On the right side it is quite impossible to make a definite diagnosis from physical examination alone, because of the normal presence of the liver dulness. The etiology and course of the illness must be taken into consideration. A skiagram may yield some information, because the serous effusion will be more transparent than the greatly bulged arch of the diaphragm. It is easier to differentiate on the left side between a sub-phrenic abscess and a simple pleural effusion, because of the extent of the dulness downwards. If the abscess contain gas, the diagnosis is easier on both sides, but on the left side the stomach note must not be mistaken for an indication of gas. The percussion over the supposed air cavity must always be compared with the true stomach note.

The most certain method of diagnosis in both groups of cases is by means of exploratory puncture. But we must not resort to it until all other diagnostic methods have been exhausted, and we are prepared to proceed immediately to radical operation. Otherwise, by puncturing through the pleural space we run the risk of making a way for the pus, which is under pressure in the sub-phrenic abscess, to open into the pleura.

If the exploratory puncture shows that pus is present, we can judge of its origin by the depth at which we reach it. A depth of several centimetres points to a sub-diaphragmatic collection, but a superficial situation is no argument against it.

One may be inclined to draw conclusions from the pressure conditions. Theoretically, pleural pus should flow out during expiration, and sub-phrenic pus during inspiration. But as a matter of fact, adhesions and indurations may also put pleural pus under pressure during inspiration. The same applies to the respiratory movements which the diaphragm imparts to the needle inserted into it.

If exploratory puncture only reveals serous fluid, this does not exclude a sub-phrenic abscess. Indeed, it strengthens our suspicion, and we must explore at a greater depth. If then we strike gas or pus, our diagnosis is confirmed, and the pleurisy is to be regarded as a neighbouring symptom dependent npon toxic irritation of the pleura.

If clinical symptoms, including pain on local pressure—a symptom we have not yet mentioned—definitely point to sub-phrenic abscess, we must not be content with one exploratory puncture which gives a negative result. We must insert the needle at one or several sittings in different directions, and thus we may at times be able to discover localized abscesses, or those difficult of access.

I have, more than once, seen the gradual disappearance of a sub-phrenic abscess which has been diagnosed from the clinical symptoms, but which the needle has failed to find. There can be no doubt that the peritoneum has the power of absorbing pus from this situation also. But if the puncture has revealed the abscess, we must not content ourselves with this possibility, but it is our duty to open it.

CHAPTER XXXVIII.

TUBERCULAR PERITONITIS.

THE surgeon has a double interest in tubercular peritonitis, which was for so long considered an exclusively medical condition. In the first place, it demands his consideration in the differentiation of the various diseases of the abdomen, and, secondly, he is often called upon to pave the way for its cure, by the performance of laparotomy. Our knowledge of the disease has been derived, to a considerable extent, from its surgery, which has also greatly increased its general interest.

Every student knows from his reading of special pathology that tuberculous peritonitis is of frequent occurrence, that it may exist in a serous, nodular or adhesive form, and that these forms may all be combined in one case. Nevertheless, it is a most common occurrence in practice to overlook these cases in their early stages. This is accounted for, to a considerable extent, by their protean nature. Many cases are diagnosed as "nervous dyspepsia," chronic, gastric, or intestinal catarrh, &c., when a careful examination of the abdomen would already reveal an effusion, or palpable tubercular masses. This oversight is due to the fact that in its early stages the disease has no specially characteristic symptoms to draw attention to its existence, and it is therefore most important to examine carefully and repeatedly patients who complain of indefinite discomfort in the abdomen.

This indefinite discomfort consists of loss of appetite, a sensation of pressure in the stomach and bowels during digestion, irregularity of the stools, occasionally diarrhœa, attacks of colic and a vague feeling of heaviness and soreness in the abdomen; sometimes also dysuria. If these symptoms have persisted for weeks or months, anæmia and emaciation supervene, and both patient and practitioner begin to think of some serious malady. If the patient is young, if he comes from a tubercular stock and has probably some previous tubercular history himself, it requires no complicated association of ideas to think of tubercular peritonitis. But in the absence of such indications, even the most experienced practitioner may grope in the dark, especially in a patient over 50 years of age.

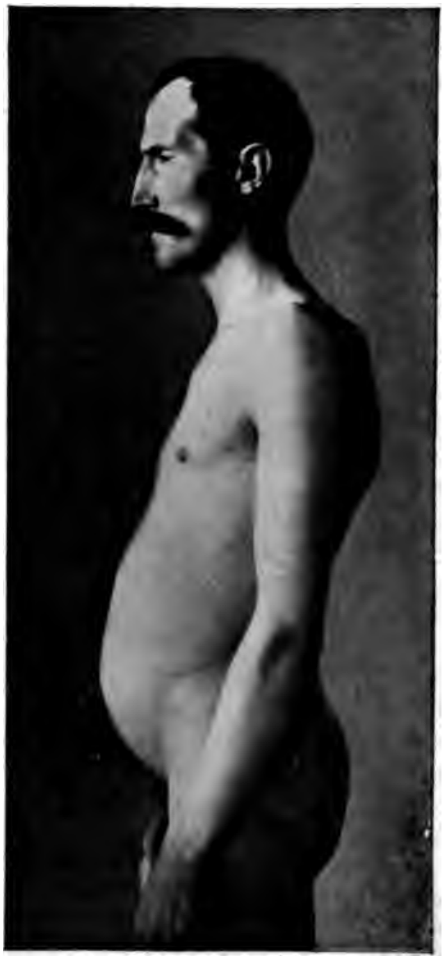


Fig. 181.—Tubercular peritonitis. Moderately extensive effusion. Abnormally prominent abdomen for a man.

After general examination, directed especially to the lungs and kidneys, in addition to any striking external signs of existing or old tuberculosis—scars or glands, bone disease—we proceed to an investigation of the abdomen. This may be quite flat, and without any abnormal dulness. But we are struck by slight rigidity of the abdominal muscles; much less than in an early septic peritonitis, but still quite demonstrable. Palpation is not really painful for the patient, but is unpleasant. Such a condition, found at repeated examinations, ought to excite serious suspicion. This represents the stage wherein the parietal peritoneum, which alone is capable of receiving sensations of pain, has become sensitive owing to the implantation of tubercles. Later on, this sensibility becomes diminished by the fluid effusion which lies as a protection between the intestine and abdominal wall, and by adhesions. This stage is common to all varieties, but its subsequent course varies considerably. In most cases, it is possible to detect a movable *effusion* after a few weeks; sometimes, however, not until after a few months. This effusion is not always abundant. If the patient when viewed in profile has an abdomen too prominent for his figure (fig. 181), if he has shifting dulness over the symphysis— N.B., when the bladder is empty — and above Poupart's ligament, this suffices to establish the presence of effusion. It is not necessary to wait until the whole abdomen fluctuates and the carriage of the patient resembles that of a pregnant woman. In other cases we may look in vain for any sign of effusion. On the contrary, some portions of the abdomen will be felt to be harder than usual, and to develop into flat, firm, cake-like masses or into roundish, and somewhat fixed *nodules*, which are nearly always painful on pressure. In certain other cases the abdomen gradually gets larger, without effusion and without these hard masses, being tympanitic all over, but remarkably incompressible and tender throughout. This is the *adhesive* form, wherein the coils of intestine are glued together in layers, by the tubercular process, thus hampering their free movement and peristaltic action.

This explains the slight compressibility of the stomach and the meteorism.

Among the *mixed forms*, it is necessary to particularize those wherein there exist *encysted effusions of fluid*, caused by a combination of adhesive and exudative processes. These effusions are generally situated in the middle or lower abdominal region, and usually contain serous fluid, but sometimes their contents are purulent, or in layers of a sero-fibrinous-purulent fluid. The nodular form of tubercular peritonitis is not as a rule pure in character, but is combined with exudative or adhesive processes.

Each of the above described varieties has its special difficulties of differential diagnosis.

(1) The purely *exudative* variety may be confused with *cirrhosis of the liver*, especially if the patient is elderly and previous addition to alcohol cannot be excluded. In such a case, an evening rise in temperature points to tubercle; but a normal temperature is no argument against it.

It should be emphasized here, that it is not enough to take the temperature now and again, at the doctor's visits. In all cases wherein tubercle is in question it must be recorded regularly for weeks, at least morning and evening. In tubercular peritonitis, as in other tubercular processes, periods of normal temperature may alternate with periods of a slight rise, or definitely high temperature.

A firm consistence of the liver, if it be palpable, and pronounced enlargement of the spleen, are points in favour of cirrhosis of the liver, whereas tenderness on slight pressure and spontaneous pains are in favour of tubercle. The presence of a pleural effusion is a strong confirmation of tubercle.

The difficulty in differential diagnosis is illustrated by the circumstance that tubercular peritonitis may lead to cirrhotic changes in the liver. According to Schönberg tubercle seems to play an important part in alcoholic cirrhosis of the liver.

Peritoneal tuberculosis may be confused with *chylous ascites*, more especially as the latter comes on as the result of tubercular swelling of the reto-peritoneal glands. It is, however, distinguished by the remarkably rapid onset of debility and the great enlargement of the abdomen, which becomes distended to an extent which is, at any rate, unusual in tubercular peritonitis. A definite diagnosis can only be obtained after exploratory puncture, which we will discuss later on.

Chronic serous peritonitis of the older authors does not enter into the problems of diagnosis. This term really includes tuberculosis, miliary carcinoma or sarcoma, and endothelioma of the peritoneum. These exceptional cases are exceedingly rare; they cannot be diagnosed even when the abdomen is opened, and can only be detected by histological or bacteriological examination.

(2) The considerations in regard to the **nodular** form are entirely different. In these cases, the diagnosis lies between tuberculosis and malignant *new growth*. To a certain extent, we should be influenced by the age of the patient; at any rate below 30 the chances are in favour of tubercle, but discretion must be exercised. After 40, the age factor has very little weight in diagnosis.

For, instance, a female, aged 60, was referred to me because of numerous abdominal tumours, which at first sight suggested cancer. Careful examination, however, showed that the case was one of nodular tubercular peritonitis, and this was confirmed by operation. On the other hand, cancer of the ovary is no rarity among women of 30 to 35 years of age.

More importance must be attached to any kind of rise in temperature, but here also caution is necessary.

A young woman, after undergoing treatment in a sanatorium for pulmonary disease, came under surgical treatment for a small, movable tumour in the right lower abdominal region. The structure was hardly as big as a walnut, but close to it were some deep, but less movable lumps, and a little free effusion was present. There were no signs of narrowing of the intestine. The elevations of the temperature were very striking: they often exceeded 102° F., and could not be explained by the condition of the lungs, in which there was nothing abnormal. Everything pointed to tubercle, but the striking mobility of the small tumour made one think of the possibility of carcinoma, because tubercular masses soon lose their mobility. Operation revealed a small carcinoma of the small intestine which had produced no constriction, and early infection of the peritoneum with carcinomatous infiltratation of the retro-peritoneal glands.

In women the clearest information can be obtained by examining the uterine appendages, which constitute the most frequent starting point both of tubercle and cancer of peritoneum. Bilateral, sausage-shaped, or nodular induration of the tubes points to tubercle or chronic gonorrhœa; the presence of a nodular tumour in the vicinity of one ovary points to cancer. But primary ovarian cancer is sometimes so small that it can hardly be detected.

The demonstration of nodules in Douglas' pouch may signify either tubercle or cancer.

(3) The purely **adhesive** forms, as far as they cause symptoms, are most likely to be mistaken for intestinal obstruction, due to chronic adhesions, which are the result of some previous inflammatory process within the abdomen. But their chronic course, the diffuse character of their symptoms, and the tenderness on pressure constitute valuable evidence for tubercle.

(4) The differential diagnosis of **encysted tubercular effusions** is of great importance, although not always easy. In these cases the question arises whether we are not dealing with a *cystic tumour—i.e.*, an ovarian, omental, or mesenteric cyst. This question is all the more pressing, because these encysted tubercular effusions often occupy the median line as we have already seen. The character of the dulness is the same in both—*i.e.*, dulness in the middle of abdomen with a normal intestinal note above and at the sides; whereas in a recent effusion the conditions are just the reverse. The decision depends upon the mobility of the structure as a whole, and it will often be necessary to give an anæsthetic in order to abolish the abdominal rigidity before determining this. A cyst, even if extensively adherent, is usually somewhat movable, and gives the impression of a round tumour, independent of the abdominal wall, if the latter is completely relaxed. An encysted effusion, on the other hand, is hardly movable at all; and even if it appears to be round in shape, is usually connected to the anterior abdominal wall.

The case of a little girl, who was suspected of having an encysted tubercular effusion, occurs to me. Careful examination showed that the structure was movable, though only slightly so, and therefore the diagnosis seemed to be a cyst, whose situation suggested an origin either from the omentum or mesentery. Operation proved that it was a large serous omental cyst, and its histological examination excluded the possibility of its tubercular origin.

Finally, it is necessary to refer to the confusion of a tubercular exudation with a sacculated *pneumococcic peritonitis*. A pneumococcic exudation causes so little surrounding inflammatory reaction that it is very often mistaken for tubercular peritonitis (fig. 177), unless the practitioner has followed the whole course of the case from the beginning. If the patient is a young girl, and there is a history of a sudden illness, starting with high fever, rigors, vomiting and diarrhœa which subsided into a quieter stage after one or two weeks, we may be quite sure that the case is one of pneumococcal peritonitis.

The "regular" course of tubercular peritonitis, as described above, is often interrupted by intervals which are caused by partial or complete intestinal obstruction, due to kinking of the bowel, by localized adhesions, or omental bands. We will again meet with these conditions, in discussing intestinal obstruction.

Our task is not exhausted with the mere diagnosis of tubercular peritonitis. We must endeavour to determine its **point of origin**, at any rate, as far as it concerns treatment. We have already mentioned tubercle of the *tubes*. A second source of origin is to be found in the *intestine*. We do not recognize intestinal tuberculosis so much by palpation as by functional disturbance, because the results of palpation in extensive tubercular peritonitis are very equivocal. Tuberculosis of the small intestine often leads to stenosis; ileo-cæcal tuberculosis nearly always does so. Tuberculosis of the large intestine usually presents the symptoms of colitis.

It must not be assumed that the association of tuberculosis of the mucous membrane of the bowel with extensive tubercular peritonitis is the regular thing. On the contrary we generally see intestinal tuberculosis combined with very localized tubercular changes in the peritoneum. The production of a widespread tubercular peritonitis probably requires the simultaneous invasion of a large amount of infective material, such as is most likely to be derived from a tuberculous tube or a softened mesenteric gland. If a tubercular ulcer of the intestine leads to tubercular peritonitis, there is usually an intermediate stage provided by tubercular glands. Tubercular peritonitis often follows a pleurisy of similar origin. As is well known, the diaphragm is not bacteria proof.

Exploratory puncture is not always harmless in cases of tubercular peritonitis, and, therefore, should only be performed when the indications are definite; for instance in the ascitic form, if the differentia diagnosis between it, cirrhosis of the liver and chylous ascites cannot otherwise be made.

It only now remains to consider how an accurate diagnosis helps us in framing indications for prognosis and treatment. That tubercular peritonitis, even in the anatomical sense, is susceptble of cure has been proved by surgical experience. Recent statistics show that one-third of the cases recover, even without operation. It has been suggested that the numerous cases which have been treated medically without any benefit, and then have promptly recovered after operation, were really on the point of spontaneous recovery. But this only begs the question and does not explain it. In the early stage, operation should only be undertaken to remove the primary disease, *e.g.*, a tuberculous tube. Otherwise we must continue for weeks, or if the patient's social circumstances permit, for months, with dietetic and climatic treatment, sunshine and X-rays. Sunshine seems to be the most important of these methods. If this is ineffectual, operation is indicated, unless there are other foci of tuberculosis which are threatening the patient's life. The recognition of the variety of tubercular peritonitis is important, therefore, not merely as a general indication, but as an element in the prognosis. This is very much more favourable in the ascitic form than in the other varieties. The prospects are rather unfavourable if the nodules undergo caseation or suppurative softening. But we should not abandon operation in these cases, because every now and again an unexpected and permanent success compensates for many failures. It is only in the purely adhesive form that operation is hopeless.

CHAPTER XXXIX.

DIAGNOSIS OF ABDOMINAL SWELLINGS IN GENERAL.

THE abdomen is the seat of various **false tumours.** Everyone knows how easily the *abdominal aorta* is felt in thin subjects, and its pseudonym "student's aneurism" is fully deserved. More than one practitioner, even, have been deceived by it. On the other hand, a genuine aneurism may be mistaken for a new growth. If the abdominal wall is not very yielding, so that the structure cannot be adequately grasped, it may be impossible to distinguish between a heaving and an expansile impulse. The phantom tumour caused by contraction of the upper part of the *abdominal rectus* is also well known. Error can generally be avoided by palpating the opposite side. If the pylorus is sensitive to pressure, the right rectus contracts when it is palpated, whereas on the left side the muscle remains quite lax. If the patient is directed to sit up without the help of his arms, it can be readily felt that the doubtful swelling is the muscle itself. The *pancreas* may constitute another false tumour. In very emaciated persons it may be distinctly felt in front of the spinal column, but it must not be mistaken for a thickened pylorus. In such emaciated persons it is quite possible to feel the pylorus and greater curvature quite separate from the pancreas—sometimes even the lesser curvature. *Fæcal accumulations* should rarely lead to mistakes, if one remembers the course of the large intestine, and if the patient has been purged before the examination. It is always important to control the physical findings by a skiagram.

In "Hirschsprung's disease" the sluggishness of the bowels may permit of the accumulation of fæces to the extent that a sarcoma is diagnosed.

Sometimes intussusception imitates a tumour, apart from the cases wherein a new growth—*e.g.*, a polypus—has caused the intussusception. The typical tumour of intussusception is recognized by its cylindrical shape and its position quite close to the spinal column.

Tumours and swellings of the abdominal wall, described in a separate chapter, must not be confused with abdominal tumours.

Finally, it must be remembered that inflammatory changes may resemble abdominal tumours. This applies to inflammatory swellings of the omentum, which develop as a result of resection of the omentum in operations for hernia, where there has been some failure of asepsis, as first shown by Braun. A clue is afforded by the history and the pyrexia.

An ordinary *appendicitis* may sometimes develop into a hard immovable lump, as large as a fist or larger, filling up the pelvic fossa, and requiring weeks or months for its absorption. This phenomenon is due either to some special peculiarity of the organisms causing the inflammation, or to some abnormally sluggish reaction of the system.

In a case of this kind, in an elderly man, even at the operation I made a provisional diagnosis of cancer of the cæcum, and performed an intestinal anastamosis. A few months later the whole resistance had disappeared, and the patient enjoyed good health for seven years after the operation.

Such cases are very similar to the "phlegmon ligneux" of the neck. If we consider the extent and duration of the induration which may be caused by a small perimetritic abscess, it is not at all surprising that a similar result may ensue from appendicitis.

Actinomycosis occasionally causes a movable swelling in the ileocæcal region, but, in contrast to cancer, it does not usually lead to symptoms of obstruction.

If a genuine—or false—abdominal tumour is definitely *movable*, its pedicle affords the best indication of its origin. But as this pedicle cannot always be felt, we can draw some conclusion as to its origin from the segment of the circle which can be described by the tumour, as pointed out by Pagenstecher. This task will be facilitated by marking the segment of the circle on the abdominal wall. For instance, if the case is one of movable swelling of the gall-bladder, wherein the tumour can be displaced even as far as the left hypochondrium, the centre of the segment of the circle described by the tumour will always be at the normal site of attachment of the gall-bladder. The curve will be concave upwards in contrast to the curve of a pedunculated ovarian cyst, which is concave downwards, with a centre which is either in the mid-line or more or less to one side. In a similar manner, it is possible to ascertain the origin of a swelling which consists of a floating kidney.

An organ which under normal conditions is only slightly movable, may be susceptible of considerable displacement when it is the seat of a tumour, especially the pylorus. For instance, I found a carcinoma of the lesser curvature, the size of a large fist, situated at the anterior superior spine of the left ilium, in a young girl; the tumour could be dragged about, almost all over the abdomen (see under Cancer of the Stomach).

Tumour formation in an organ which is congenitally displaced may render the diagnosis difficult. This applies particularly to the kidneys, which may be found lying transversely in front of the spine, or more laterally at the inlet of the true pelvis.

It has occasionally been noted that an ovarian cyst may become free and take up its quarters elsewhere. This of course causes an insoluble problem in diagnosis, before the abdomen is opened. On one occasion, I found a left ovarian dermoid engrafted on the hepatic flexure of the colon. On another occasion it was situated in the middle of the abdomen, enveloped by omentum. The remains of the tube were still attached to the left side of the uterus, in both cases.

If a swelling is remarkably movable, but no indication is afforded by its pedicle or by the curve described by its movement, we should think of a tumour of the *small intestine, mesentery or omentum*, if it is situated in the middle of the abdomen. The first is the most probable if the tumour is hard or uneven; the other two if it is roundish and elastic in consistence.

The determination of the point of origin is easier if the tumour is *less movable* and *not too large*, because the number of organs with which it might be connected are more limited. But even then, it may be difficult to diagnose between cancer of the duodenum and of the pancreas, enlarged gall-bladder and a commencing hydronephrosis, cancer of the intestine and a displaced kidney which has become fixed, &c. A correct diagnosis can only be formed by taking into consideration at the same time the history, the functional disturbance, the condition found on palpation, and a skiagram.

Tumours which occupy the *whole* or almost the whole of the abdominal cavity are particularly difficult to diagnose. If they are *hard* and the enormous leukæmic spleen can be excluded, they are essentially *fibro-myomata of the uterus*, rarely *fibro-sarcomata* of the ovary. Fibromata or fibro-myxo-lipomata of the fatty capsule of the kidney are also possible. In children they are usually enormous *sarcomata* or *mixed tumours of the kidneys*. Bimanual gynæcological examination, and if necessary the sounding of the uterus, may afford a conclusive decision. A sarcoma of the kidney is recognized by its somewhat lateral position, if it has become very extensive, and by the fact that it reaches up into the hypochondrium. Tumours of the fatty capsule of the kidney, just referred to, do not adhere to any rule.

If the tumour is softly elastic, or fluctuating and therefore probably a cyst, it is necessary to distinguish between *ovarian cyst* and ***hydronephrosis***. The former should be diagnosed if its consistence is unequal, being uneven and hard in some places, soft or tensely elastic in others. The difficulty in diagnosis really begins when the cystic tumours are uniform.

An ovarian tumour of moderate size can be best defined at its superior border, a hydronephrosis at its lower border. In more advanced stages, external examination may be quite inconclusive. If both ovaries can be felt on bimanual examination, the matter is decided, because parovarian cysts are not in question where large tumours are concerned. But if, as usually happens, the two ovaries cannot be felt, the large intestine should be distended to see whether the bowel lies to the other side of and above the tumour, or to its inner side and beneath it. In the latter case it is certainly a renal tumour; in the former, probably an ovarian tumour. We say "probably," because we have also seen the large intestine running above and to the outer side of a renal tumour. But if this proceeding leads to no definite conclusion, we are thrown back on a consideration of the previous history. In the case of hydronephrosis there will probably have been attacks of renal colic with sudden and profuse micturition of clear or blood-stained urine. The patient will also have noticed that the tumour which before had been situated high up has now gradually become lower. An ovarian tumour, on the other hand, is practically painless, unless subjected to torsion occasionally, and grows from below upwards. Finally, a decisive conclusion may be arrived at by cystoscopy. If urine escapes from one ureter only, we have every reason for assuming that there is a closed hydronephrosis on the other side. If both ureters are functional we must exclude hydronephrosis.

If the tumour does not fit in any of these categories, we should think of one of the rare *mesenteric omental cysts* (the latter, as experience shows, especially in young girls), or an *encapsuled tubercular peritonitis*, if the structure seems to be less movable and less well defined than a genuine cyst.

The rare condition of *cystoma of the uterus* is very apt to cause error. In one such case, the tumour filled up both hypochondria, although the cervix was prolapsed between the labia.

We must also think of the *pregnant uterus* which has sometimes been subjected to the surgeon's knife, to the silent but malicious satisfaction of the accoucheur. It is therefore by no means superfluous to examine the breasts in doubtful cases, and also to employ the stethoscope. It is of course well known that too much reliance cannot be placed on the history, in these circumstances. If in a case of suspected pregnancy the size of the uterus is too large for its estimated duration, and if no fœtal parts are felt, we should think of a *hydatidiform mole*. If, in a case of suspected pregnancy, a slightly movable or a definitely movable tumour is found at the side of a somewhat enlarged and soft uterus, we should diagnose an extra-uterine gestation.

Every tumour raises the question of **innocence** or **malignancy**; a question which we can very often answer, even if the origin of the tumour is not clear. Three signs indicate malignancy: (1) Rapid growth, and early emaciation of the patient; (2) the presence of free fluid in the abdominal cavity; and (3) multiplicity of the new growth.

To demonstrate a *small* amount of *effusion*, it is necessary to percuss as *gently as possible*, with the patient on the back and on the sides.

If the onset of effusion has been preceded by an exacerbation of acute peritoneal symptoms, we should always think of the possibility of torsion of an innocent ovarian tumour, more especially a dermoid. Multiplicity of the tumours indicates either malignancy or tubercle. We have already discussed their differential diagnosis, but there may be some difficulty in distinguishing them from subserous myomata.

Cancerous infiltration of the umbilicus may occasionally indicate the nature of the abdominal tumour.

As in the case of other abdominal diseases we have purposely refrained from mentioning *exploratory puncture*. Its value is still always *over-estimated*, its disadvantages *under-estimated*. Puncture of a solid tumour is harmless, unless the bowel is perforated by too powerful a needle, but it is quite useless. If we succeed in puncturing a cyst with an adequately strong needle, we must inevitably permit the escape of some of the contents of the cyst into the abdominal cavity. This may be a matter of indifference if the swelling is innocent, but it is most undesirable in cases of cancer, hydatid or suppurating cyst. Thus I was once consulted in the case of a young girl who developed acute peritonitis as a consequence of exploratory puncture of a cystic abdominal swelling. Operation was performed immediately, and it revealed a suppurating ovarian cyst in which the puncture was still visible as the starting point of the suppurative peritonitis. As every ovarian cyst, and every tumour whose differential diagnosis is doubtful should be operated on, and as exploratory puncture so often leaves us in the lurch, this procedure is limited in practical value to the information which it may give as to the aseptic or purulent condition of the contents of the cyst. But, as just stated, puncture is least permissible if there be any suspicion of suppuration. If, however, we consider puncture to be indispensable, it should not be done until everything is in readiness for operation, or better still until after the abdomen has been opened and the cyst exposed. We can then, at any rate, prevent the unnoticed entrance of pus into the abdominal cavity.

CHAPTER XL.

A REVIEW OF THE SURGICAL DISEASES OF THE STOMACH AND DUODENUM.

ALTHOUGH we have ceased operating for functional diseases of the stomach, surgery still maintains an important position in the treatment of chronic gastric disease, because of the frequency of gastric ulcer. It therefore behoves the surgeon to be familiar with the diagnosis of gastric disorders, in order to recognize the cases which come within his domain. This task is greatly facilitated by the work of Rieder and others who have developed gastric skiagraphy, and have enabled us to put to the test of direct vision the indirect diagnostic conclusions with which we have hitherto been content. Other methods of diagnosis must, however, not be neglected, lest we merely substitute modern mistakes for ancient errors.

Our first endeavour, in dealing with a gastric patient is to determine whether the case is medical or surgical. This can be ascertained by a well-directed investigation of the four symptoms which suggest to the patient that he is suffering from his stomach, and compel him to seek advice. These symptoms are : (1) loss of appetite, (2) pain, (3) motor derangements, and (4) hæmorrhage.

(1) **Loss of Appetite.**—This symptom is so indefinite, that one must be extremely careful in drawing any conclusion therefrom. It is a symptom common to smokers, alcoholic and kidney patients. All severe general diseases are associated with a diminution of nutritive requirements. Victims of neurasthenia and hysteria complain of loss of appetite, even when their well-nourished appearance belies their complaint. This symptom only possesses surgical importance, if all other causes can be excluded, and if it has come on at a definite time, in a patient whose stomach has hitherto been quite healthy. It may then be an important early sign of cancer of the stomach. But otherwise, we should merely take note of the symptom, without, however, allowing it to influence our diagnosis in any way.

(2) **Pain.**—Three varieties of pain occur in the region of the stomach.

(*a*) *Persistent pain.* A pain in the neighbourhood of the stomach which is persistent, or does not cease completely, is always a sign of serious disease. It may signify extensive perigastric adhesions resulting from gastric ulcer, pancreatic inflammation, or disease of the biliary passages. In these cases, the trouble will usually have arisen at some definite point of time, and will have developed into a perigastric abscess, or into an inflamed cyst of the pancreas, &c.,

conditions which are generally more or less apppeciable. But if the beginning of the illness has been indefinite, we should think of a malignant neoplasm—although no swelling may be palpable—which has involved the retro-peritoneal tissue. These cases are usually cancer of the body of the stomach, but occasionally cancer of the pancreas or a primary growth of the liver.

(*b*) *Intermittent pain occurring at some definite time of day.* Sometimes this is simply functional, *i.e.*, *gastralgia*. It is distinguished from gall-bladder pain by its tendency to radiate towards the *left* side of the back or shoulder, and by the greater regularity of its onset. This pain is often due to some functional derangement without ascertainable cause. Excessive smoking may be the cause, in which case it is not merely the quantity and quality of the tobacco which are at fault, for the sensitiveness of the smoker must also be taken into account. But this pain is often a symptom of gastric ulcer, and this is particularly likely if the pain has some regular relation to meals.

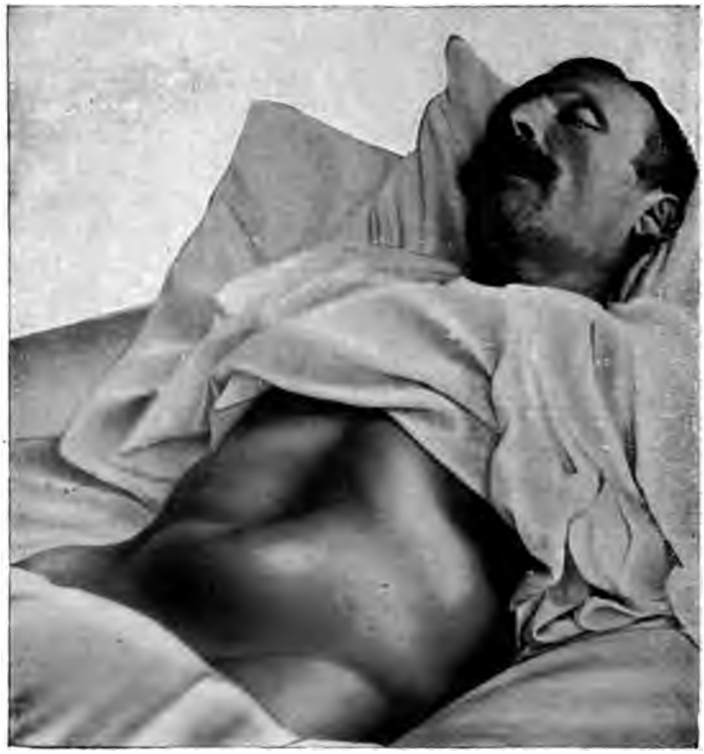


FIG. 182.—Stenosis of the pylorus due to cancer. An attack of gastric rigidity. (Stomach not artificially distended.)

We shall discuss later on the significance of the amount of interval between the meal and the pain. This characteristic pain of gastric ulcer must be distinguished from painful contraction of the stomach in stenosis, which will be referred to immediately.

(*c*) *Intensely violent pain in the epigastrium, of sudden onset.*—If the pain is definitely situated on the right side, it nearly always indicates gall-stone colic, as we shall see later on. If it is situated towards the middle line or towards the left, it constitutes one of the chief signs of perforation of the stomach.

(3) **Motor disturbances.**—If the patient complains of unaccustomed pressure in the gastric region, after meals, or of painful distension recurring at short intervals during digestion, and if we detect palpable and visible signs of gastric contractions during the attacks, we must assume that there is some narrowing of the pylorus. Vomiting is by no means a necessary concomitant of this condition, because patients are able to avoid this symptom, even in severe stenosis, by a careful choice of diet. Bnt if vomiting be present, it will exhibit the classical sign of retention vomiting, *i.e.*, the expulsion of large amounts of food residue, taken one or two or more days previously, in a more or less fermenting condition. This vomiting is especially frequent in the morning. It is to be distinguished from the morning sickness of alcoholism, which merely evacutes mucus ; from the morning vomiting of certain neurasthenics who bring up gastric juice which contains hydrochloric acid ; and more particularly from hysterical vomiting, in which condition the stomach is often emptied immediately after a meal.

The following case is important in this connection. A young girl had suffered from constant vomiting for some years, and it had therefore been necessary to treat her carefully with rectal and subcutaneous feeding. The patient and her parents had decided upon a "curative" operation, and I was called in to perform a gastro-enterostomy. The history showed that the vomiting always came on immediately after meals, and investigation proved that the chemical and motor functions of the stomach were normal. The removal of the patient from her surroundings, and the peremptory prohibition of vomiting worked wonders. The vomiting ceased forthwith ; all food was kept down and the body weight increased by 12 kilogrammes in a few weeks. The patient has remained well for some years.

Unfortunately similar cases have often been submitted to operation, because the surgeon has made a wrong diagnosis, or has not appreciated the power of moral treatment. If the latter fails there is no prospect of permanent benefit from any operation. Surgery must not be resorted to without a previous exhaustive examination of the stomach and of the entire patient ; but this should also be the antecedent to any form of psycho-therapy.

There is another form of vomiting which may lead us astray, which is due to a diffuse fibrosis of the wall of the stomach, resulting from cancerous degeneration of the mucous membrane. This vomiting does not partake of the character of retention vomiting, but comes on directly after a meal like hysterical vomiting.

The reader is reminded that functional or organic stenosis of the lower segment of a dilated œsophagus, leads to regurgitation of food, but this must not be mistaken for true vomiting.

If the clinical signs of retention are present we may be confident of the diagnosis of stenosis of the pylorus. It is however more difficult to determine the character of this stenosis. Even if there be a history of stomach trouble extending over many years and indicating that the stenosis is due to ulcer, we are unable to decide whether cancer may be developing in the scar or in the still existent ulcer—a matter which will be dealt with later on.

(4) **Hæmorrhages.**—If the patient complains of hæmorrhage, or if we are called in at the actual time of bleeding, we must first decide that the blood is really coming from the stomach. Hæmorrhage due to pulmonary tuberculosis must be excluded. In cirrhosis of the liver there may be bleeding from a varicose dilation of the œsophageal veins. Hysterical patients are able to extract blood from their gums and expel it mixed with saliva; or they may swallow it first and vomit it with the contents of the stomach. I am convinced that it is sometimes very difficult to distinguish such bleeding from true gastric hæmorrhage, and I have myself in company with the attending physician been victimized by a hysterical patient. But as Josserand pointed out, this kind of hæmorrhage is characterized by an abundant admixture of mucus, so that the vomit resembles fruit juice and does not usually coagulate.

If the patient does not complain of vomiting blood we must enquire whether there is any blood in the stools. If they contain blood the patient will usually describe them as looking like tar. We must make sure that this appearance is not due to bismuth, for it is very likely that the patient may have been taking this drug for his gastric symptoms.

If the hæmorrhage is arterial in character, or if it is profuse in quantity, experience shows that it is probably due to an ulcer. If it appears in the vomit like dark lumps of coffee grounds, we must assume that cancer is present. There are indeed exceptions to this old rule, but it is an excellent guide to diagnosis.

Before we proceed to a more detailed examination of the patient we must extend our study of the case by the consideration of a few *general points.*

We have already seen, when discussing loss of appetite, that the patients are divisible into two groups. There are those whose symptoms are of recent origin, the patients stating that they had hitherto been endowed with the digestion of an ostrich, and there are those who have been chronic sufferers. In regard to the former group, the question of carcinoma is the first to occur.

The patients who have been suffering from their stomachs for very many years are themselves divisible into two classes. The one class adheres strictly to a "diet," as prescribed by a specialist or a nature-healer; but if occasion requires, these patients are quite equal to a sumptuous meal. They often come to consult a surgeon, after reading some popular article on medicine, which has made them apprehensive of cancer, although their symptoms may not have become any worse. Of course, it is quite possible that this class of patient may be affected with some organic disease; but in the absence of any evidence thereof, we must persuade the patient to live like a normal being. If our persuasion fails, we must leave the patient to his own particular dietary system.

The patients of the other class, with their previous history, which includes severe hæmorrhages, obviously are affected with gastric ulcer. Their symptoms may be due to an old unhealed ulcer, or to the formation of new ones. Many of these patients, despite the evidence of organic disease which they bear, have undergone considerable treatment for their nerves, and indeed their nervous system may have suffered in consequence of their disease. The effect of gastric disease upon the psychical condition is well known. It gives rise to a special form of mental "fatigue," which is quite characteristic as the result of the pain of an old-standing gastric ulcer.

So far for the *history*. We now proceed to *physical examination*, beginning with *inspection*.

A striking pallor, and a hæmoglobin content of less than 50 per cent. suggest the presence of an ulcer, if those symptoms have been preceded by a hæmorrhage; if not, the case is very probably cancer. This latter diagnosis becomes a practical certainty, if jaundice is also present; but in this event the case is no longer operable. This remark also applies in the presence of cancerous glands above the left clavicle.

We now proceed to *palpation*. If this manipulation elicits the signs of gastric contraction, it is quite certain that pyloric stenosis exists. If a tumour is felt in the pyloric region, a carcinoma is probably present; but this tumour may also be due to an old indolent ulcer which has produced inflammatory induration in the adjacent stomach wall, the gastric musculature hypertrophying from overwork. These inflammatory lumps rarely exceed the size of a hen's egg. Their surface is usually smooth, their mobility not very great; in shape they resemble an obtuse cone with the base towards the left. If they are nodular in shape, or if other independent swellings are also present, cancer is indicated.

If the swelling is in the middle line, at the level of the lesser curvature, or more towards the left in the neighbourhood of the greater curvature, it is almost certainly a carcinoma, because palpable swellings due to ulcer are very rare in this locality. Tumours of the transverse colon are usually recognized as such, with ease, after careful palpation ; but it is easy to mistake cancer of the pancreas for cancer of the stomach. Tumours of the spleen, and even of the kidney may occasionally give rise to doubt. Sometimes the slightest pressure upon the epigastrium will elicit reflex muscular contraction, pointing to the existence of organic disease. If palpation reveals nothing abnormal, the diagnosis of ulcer or of cancer must be left an open question, because most ulcers and a large proportion of cancers yield nothing to palpation. We should search the rest of the abdomen for the presence of metastases which can be felt, or for fluid effusion. Either of these conditions indicate malignant new growth. Sometimes a secondary nodule develops early in Douglas' pouch, and can be detected on rectal examination.

We now proceed to the clinical investigation of *retention*, and the examination of the *chemistry* of the stomach.

This is not the place for a detailed exposition of the numerous clinical methods of testing gastric mobility, or examining the conditions of the secretions. Their complexity is very often much greater than their clinical value. Many of these methods are superseded by an X-ray examination. The following procedure will be found adequate in practice.

The patient is given a meal in the evening, consisting of food which is easily recognizable—a dozen stewed plums for preference. On the following morning—*i.e.*, twelve hours subsequently—the stomach contents are syphoned off. If the viscus is empty, there is obviously no retention. If there be much mucus some gastric catarrh exists, possibly of an alcoholic nature. If we obtain gastric juice with hydrochloric acid, there is clearly excessive secretion going on which may be purely functional, but which must arouse the suspicion of ulcer. If we find any of the plums of the preceding evening, or may be food residue of previous days, this constitutes clinical evidence of retention, the degree of which is estimated from the amount of fluid syphoned off. After the stomach has been carefully emptied the test breakfast of Ewald-Boas is given. This consists of a slice of dry bread and 300 cc. of tea without sugar. The stomach contents are syphoned off in three-quarters of an hour and examined for total acidity, for free hydrochloric acid or hydrochloric acid deficiency, for blood, lactic acid, lactic acid bacilli and sarcinæ. If there be any suspicion of organic disease, Glitzuisky's test may be applied, according to Kocher's modification. The stomach is washed out after syphoning off the test breakfast, and another test meal is given consisting of 100 grms. of minced meat, 15 grms. of potatoes, and 20 grms. of fat. The stomach contents are again syphoned off in two hours, and again examined in the manner just stated. If the result of the examination is uncertain the procedure must be repeated.

The chemical tests enable the following conclusions to be formed :—

(1) A high hydrochloric acid content supports the diagnosis of ulcer, but does not prove it. It may exceptionally occur in carcinoma, especially when a small pyloric cancer is present, causing stenosis.

(2) The absence of free hydrochloric acid, or the presence of a hydrochloric acid deficiency supports the diagnosis of carcinoma, but occurs frequently as a purely functional disturbance of secretion.

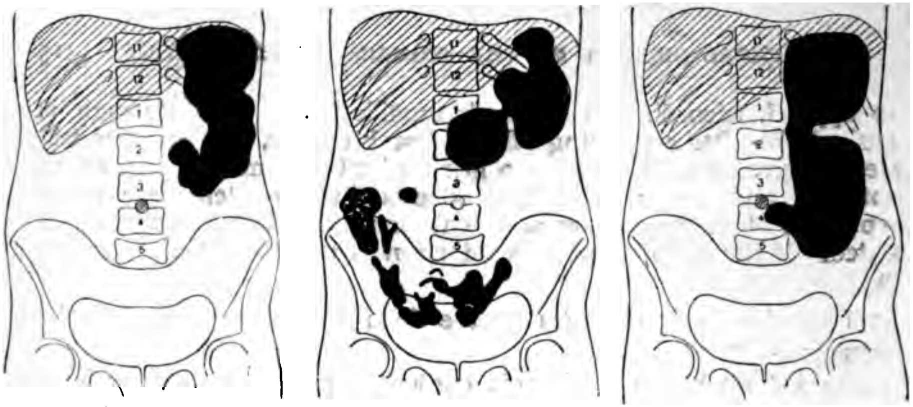
(3) The absence of lactic acid is not evidence against cancer, but if definitely present it constitutes a strong suspicion of cancer. The same applies to the absence or presence of the *Bacillus longus*.

(4) A greater secretion of hydrochloric acid after the test meal, than after the test breakfast, indicates either a healthy stomach, or an ulcer. A decrease of hydrochloric acid secretion after the test meal indicates cancer, but it may also occur in other debilitating diseases.

(5) The presence of blood generally indicates some ulcerative process, assuming that the stomach has been emptied carefully with a soft tube, and no injury has been done to the mucous membrane of the throat. But as it is not always possible to exclude the risk of injury to the mucous membrane by the passage of the tube, it is indispensable that doubtful cases should have the stools examined repeatedly for the chemical detection of traces of blood after two days' abstinence from meat food. The more constantly blood is detected, the more suggestive of cancer is the case ; because the admixture with blood is very irregular in cases of ulcer.

This clinical investigation will have afforded sufficient information about the case to enable the best use to be made of *X-ray examination.*

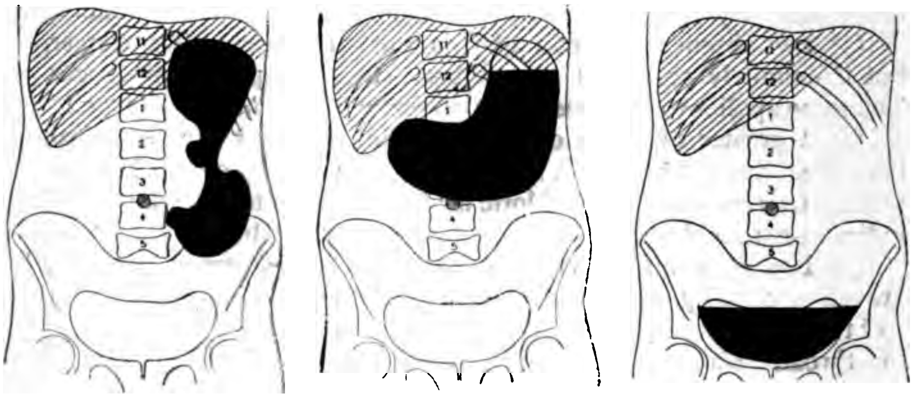
A definite method must be pursued in order to obtain reliable results. As a preliminary we must get rid of the idea, which is prevalent in some quarters, that skiagraphy can portray for us, in some magical manner, the state of the entire gastro-intestinal canal, on one picture. Some cases do indeed permit a diagnosis to be made from the first plate, but most cases require a series of three or four plates to yield any definite conclusion. Sometimes it will be necessary to repeat the whole series; or supplementary plates may have to be taken at special times. In very many cases it is important to follow the course of the *entire digestive process*, and the behaviour of the



(*a*)
Peristaltic restlessness of the stomach in tabes.

(*b*)
Abnormally severe peristalsis in a case of innocent pyloric stenosis.

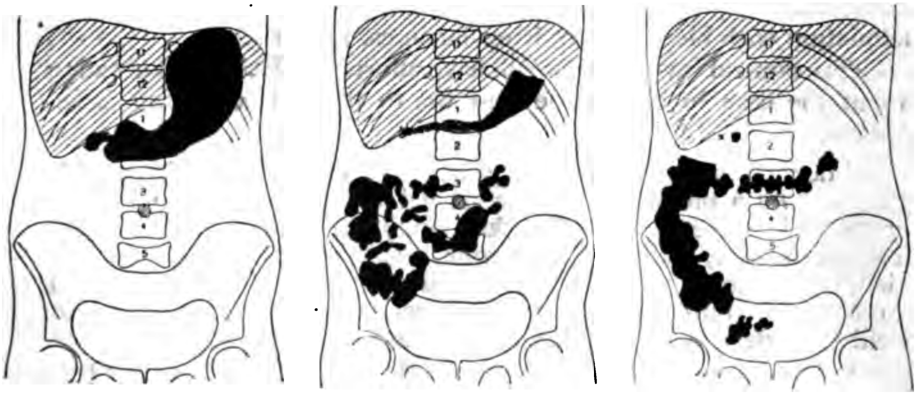
(*c*)
Small, flat ulcer at the lesser curvature, with spastic contraction of the greater curvature.



(*d*)
Cicatricial hour-glass contraction of stomach with deep ulcer of the lesser curvature (Haudek's notch).

(*e*)
Cicatricial stenosis of the pylorus with great distension of stomach, in its breadth.

(*f*)
Innocent pyloric stenosis showing "soup plate" residue after twenty-four hours.

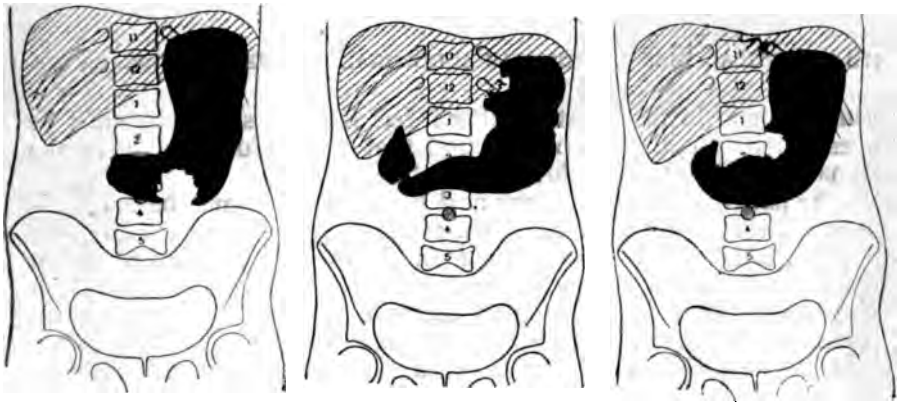


(*g*)
Duodenal ulcer, plate taken immediately. Rapid emptying.

(*h*)
The same after two hours. Stomach nearly empty.

(*i*)
The same after six hours. Stomach empty. × duodenal stain.

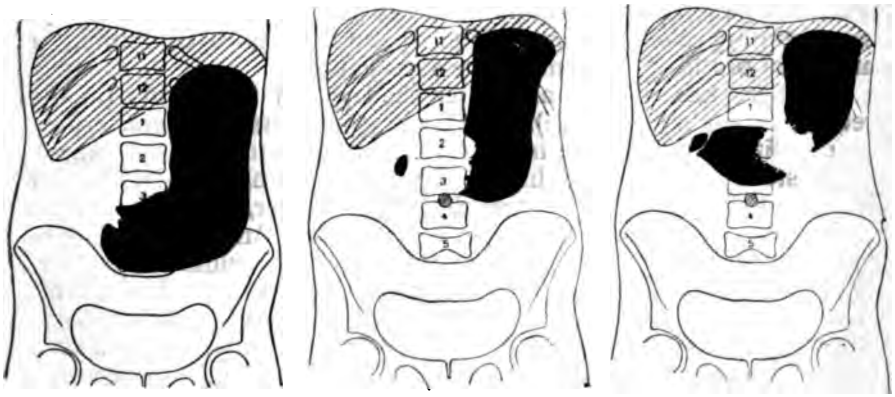
FIG. 183.—Semi-diagrammatic illustrations of skiagrams of the stomach. (See also fig. 175.)



(*k*)
Long shadow due to cancer of greater curvature.

(*l*)
Cancer of cardiac end of stomach. (Trendelenburg's position.)

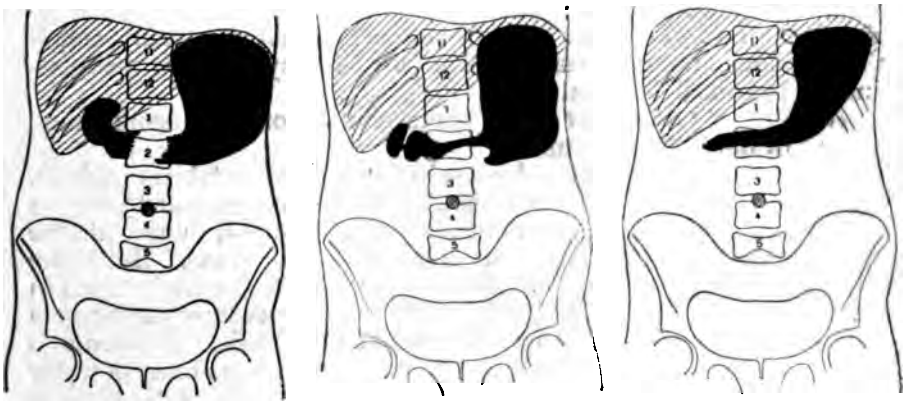
(*m*)
Loss of shadow due to cancer of lesser curvature.



(*n*)
Cancer of pylorus. Its duodenal end badly defined instead of the normal clear definition. (Compare 175 *h* and *c*, and 183 *e*.)

(*o*)
Cancerous degeneration of antrum pylori, with its complete absence from the shadow.

(*p*)
Cancerous hour-glass stomach.



(*q*)
Loss of shadow through pressure of vertebra. (Abdominal position.)

(*r*)
Loss of shadow through a tumour, outside the stomach.

(*s*)
Shrinking of the stomach in diffuse scirrhus (funnel-shaped).

FIG. 183.—Semi-diagrammatic illustrations of skiagrams of the stomach. (See also fig. 175.)

bismuth meal in the intestine, after it has left the stomach, must be investigated. It may then be found that the lesion is not in the stomach at all, but lower down in the bowel.

The patient, whose stomach and large intestine must be empty, is given a meal consisting of 40 grms. of bismuth carbonate (or 80 grms. of chemically pure barium-sulphate) and 400-600 grms. of thin carbohydrate porridge (without albumen or fat, therefore no milk). A skiagram is taken immediately in the erect and recumbent postures (plate on abdomen). In some cases an additional skiagram in the right lateral position will be required. These pictures will indicate the shape of the stomach, and will frequently show a loss of shadow due to the presence of cancer, or a notch due to ulcer. If the skiagram appears to be normal, and nevertheless there be good clinical reasons for suspecting organic disease, another plate must be taken in the Trendelenburg position, because this is the only possible way of demonstrating ulcers situated high up on the lesser curvature, or carcinoma of the cardiac end of the stomach.

Another plate must be taken after the lapse of two hours (in the erect or recumbent posture). This will give information concerning the motility of the stomach. In normal conditions, the stomach should still contain at least half of the bismuth meal, but if the motor power of the stomach be increased (duodenal ulcer, carcinoma of the fundus) it may be nearly or completely empty. Another plate is to be taken after six hours, either in the erect or recumbent posture, and the patient must abstain from liquid and solid food until this time. The stomach will then be completely empty in normal conditions, but small particles of residue will occasionally be found even in healthy persons, especially if there be any severe degree of displacement. A residue of less than a half of the bismuth meal will be found after 6 hours in hyperacidity without ulcer, in toxic or reflex pyloric spasm (*e.g.*, in ulcer of the lesser curvature) and in some cases of duodenal ulcer. A greater residue than this, after 6 hours, indicates functional stenosis, due to an ulcer at the pylorus, or is suspicious of organic stricture thereof. In all such cases, another skiagram must be taken in 12-24 hours. If the stomach is not absolutely empty after 24 hours, the diagnosis of stenosis of the pylorus—or the intestine—is practically certain.

A skiagram taken after 24-48 hours gives information concerning the rest of the intestinal canal.

We have hitherto confined ourselves to skiagrams taken on plates, because the surgeon does not make the skiagraphic examinations personally, and depends upon the radiologist to supply him with the pictures upon which he bases his diagnostic conclusions. But if the surgeon has the time and opportunity to make screen examinations in the intervals of the taking of the plates, this procedure will be very advantageous, especially if the character of the gastric movements is of importance for the diagnosis. Screen examinations must however not occupy much time immediately after the bismuth meal, because it is necessary that the first plate should be taken with as little delay as possible.

It is necessary to be acquainted with the skiagram of the normal stomach, in order to draw correct inferences from skiagrams of abnormal stomachs. Fig. 175 represents semi-diagrammatically the most important characters of the normal viscus. The effect of peristaltic movements on the shape of the stomach shadow is shown, as also the effect of adjoining anomalies. Deeply indented peristaltic waves may produce a resemblance to hour-glass contraction ; a projection between the waves may resemble a notch due to ulcer, whereas encroachments by tumours or enlarged adjacent viscera or even by distended bowel may produce loss of shadow like organic disease.

For a correct appreciation of the skiagram, it is necessary to bear in mind that the clinical signs of retention do not always coincide with the skiagraphic appearances. A stomach may empty itself of the bismuth meal within six hours, and yet contain a projecting carcinoma or a cicatrical stenosis which would keep back more solid food for days. In a case of pyloric cancer, wherein the stomach emptied itself of the bismuth meal after six hours, I found on operating, that a dozen undigested figs had been retained in it for fourteen days. The length of time which the bismuth meal remains in the stomach, depends to a great extent upon its composition. Our own conclusions are based upon observations made with pure bismuth and carbohydrate porridge with the addition of a little cocoa. If the bismuth meal contains much albuminous or fatty material, it remains considerably longer in the stomach. On the other hand a mere bismuth emulsion leaves the stomach more quickly than the carbohydrate porridge, and pure water more quickly than the emulsion. This point has a definite diagnostic bearing, as we shall see later on.

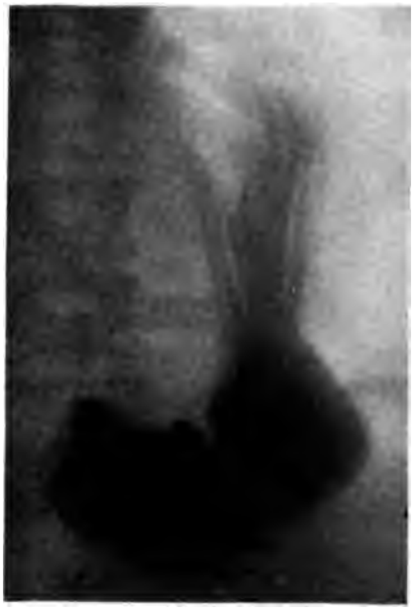


FIG. 184. — Longitudinal folds of the gastric mucous membrane. Encroachment of the spleen on to shadow of stomach.

To summarize the diagnostic conclusions which may be drawn from a skiagram, with due consideration to the foregoing remarks, it may be stated that :—

(1) **Ulcer of the fundus of the stomach** is sometimes quite unrecognizable, but frequently is revealed by a persistent spasm at the level of the ulcer, or a notch-like projection of the stomach (Haudek's notch).

(2) **Cancer of the fundus of the stomach** is usually revealed by irregular losses of shadow in the skiagram.

(3) **Cicatricial or cancerous stenosis of the pylorus** is recognized by dilatation of the stomach in its breadth, and by food retention for twelve to twenty-four hours.

We now have sufficient information about our patient to enable us to classify him in one of the main categories of surgical gastric disease, viz., displacement, ulcer or new growth. We will proceed to deal with these in detail after a few remarks on foreign bodies.

A.—FOREIGN BODIES IN THE STOMACH.

It is well known that a variety of articles may gain access to the stomachs of jugglers and the insane. As a rule, even large objects, such as spoons, forks, thermometers, &c., pass on spontaneously, and find their way through the much narrower intestinal canal, without injuring it. Surgical intervention is, therefore, but rarely required. Should it, however, be necessary, there is no difficulty about the diagnosis, and if the history is unreliable a skiagram will generally elucidate any obscurity. It is noteworthy that small foreign bodies are more likely to remain in the stomach than larger ones.

In proof of this it is only necessary to refer to the case in which the whole contents of a nail-box were removed from the stomach: over 1,500 nails, hooks, tacks, conglomerated into a mass weighing 1 kilo, certainly constitute a record, even if similar cases have been described more than once.

We may refer here to **concretions** which develop in rare instances, within the stomach itself. If we feel a strikingly movable hard lump through the abdominal wall, and the patient is a man who has much to do with varnish, and is addicted to strong drink, we should think of a *resin stone*. If such a swelling is found in a girl with a long plait, who confesses to biting her hair frequently, the diagnosis is obviously a *hair tumour*.

B.—DISPLACEMENTS OF THE STOMACH.

We have referred to these, briefly, in discussing the abnormalities in position of the abdominal viscera. These displacements possess two distinctive signs: (1) The low level of the greater curvature, sometimes even as low as the symphysis, easily demonstrable by ordinary clinical methods; and (2) the low level of the pylorus, which can often be demonstrated by palpation, but quite easily by a skiagram. This does not, however, convert the case into a surgical one. A displaced stomach so often discharges its functions quite normally, that we must rather attribute the main cause of symptoms to inefficient muscular power, or to inefficient—or more correctly to purposeless—innervation, and not to the mechanical circumstance of the gastric ptosis. Even if it has been proved clinically, and where possible also by a skiagram, that the stomach does not empty itself quickly enough, *i.e.*, in the course of six hours, this is not sufficient to justify operation, if it is the only symptom present.

Operative fixation of the stomach, which in its ultimate results is always a doubtful procedure, although occasionally useful, should only be considered in case of pronounced retention and profound disturbance of the general nutrition.

It may be mentioned as a curiosity that the stomach has often been found twisted, with the circulatory disturbances corresponding to a volvulus. This condition has not yet been diagnosed, but it should be suggested by fruitless movements of vomiting and by the impossibility of emptying the distended stomach by means of the tube.

C.—GASTRIC AND DUODENAL ULCER.

We suspect gastric ulcer clinically as already stated, if pain comes on regularly during digestion, if there is tenderness on pressure over the lesser curvature, the pylorus or duodenum, and if there is excess of hydrochloric acid. The diagnosis is rendered certain by severe hæmorrhage—hæmatemesis or melæna—or by the association of the above symptoms with frequent traces of blood in the stools. Three questions then arise, which concern the site of the ulcer, the actual pathological changes which have occurred and the possibility of cancerous degeneration.

(1) THE SITE OF THE ULCER.

Clinical investigation will supply the following points. If the pain occurs in the first half of the digestive period the ulcer is usually in the stomach itself. If the pain occurs in the second half of the digestive period—hunger-pain—the ulcer is situated in the neighbourhood of the pylorus or on its duodenal side. In these cases the evening meal is often followed by pain in the middle of the night. There are, however, so many exceptions to this rule, that we must not jump to the conclusion that hunger-pain always means a duodenal ulcer. It has, indeed, been shown that this pain may exist without the slightest trace of ulcer.

In ulcer of the stomach the pain on pressure is usually on the left and radiates towards the left side of the back and the left shoulder, whereas the position of duodenal pain is more towards the right.

The persistence of symptoms of ulcer for many years without retention, indicates either gastric or duodenal ulcer; but the gradual onset of retention would point to pyloric ulcer, or possibly the development of hour-glass stomach.

The chemistry of the stomach is on the whole of little assistance. Gastric ulcer is usually associated with hyper-acidity. In duodenal ulcer, the acidity may be excessive, deficient or normal. If blood is found in the stomach contents, and in the stools, it points to gastric ulcer, but if found in the stools only, and persistently, the case is one of duodenal ulcer.

The clinical indications on the whole are not very conclusive, and until recent years the diagnosis of the position of the ulcers was very uncertain. The assistance which is now afforded by X-ray examination is, therefore, of the utmost value. Gastric ulcers are with few exceptions only visible when they reveal themselves on the outline of the gastric shadow. Superficial ulcers are therefore invisible. As soon as they reach the muscular layer they appear in the gastric shadow as sharply circumscribed projections which, viewed from the inside of the stomach would look like depressions. If the ulcer has penetrated the wall of the stomach and has extended into the gastro-hepatic ligament, or into the liver or pancreas, or more rarely the spleen, the skiagram will reveal a projection of the gastric shadow more or less filled with the bismuth meal; or it may have the appearance of notches whose contents, in erect patients, are often superimposed by a small air vesicle. The notches in the vicinity of the lesser curvature get into the border of the gastric shadow and become visible in the skiagram, because the adhesions around these notches act as fixed points for the stomach whence the viscus tilts backwards or forwards, corresponding to the anterior or posterior position of the ulcer. Beginners are apt to mistake for these notches the shadows of the transverse processes or calcareous glands if they happen to lie on the margin of the gastric shadow.

Occasionally, irregular spastic contraction of the stomach wall may cause projecting diverticula, and these look just like Haudek's notches on the skiagram, being also superimposed by the air vesicle—but the condition is very rare.

A frequent but not invariable appearance is a sharply defined spastic contraction of the greater curvature opposite the ulcer, suggesting a resemblance, on the skiagram, to an hour-glass stomach, even if the lumen of the stomach remains quite wide. The true hour-glass stomach, due to cicatricial stenosis, is easily distinguished from this spastic contraction, by its double-funnel shape, and by the greater length of the junction between the two halves. There are, however, intermediate forms, wherein spastic contraction is associated with a moderate cicatricial stenosis. In these cases the diagnosis is indefinite. The diagnosis of ulcer of the lesser curvature is confirmed by the delayed emptying of the lower segment of the stomach, due either to reflex spasm, or cicatricial distortion of the intact pylorus, or to a combination of both causes.

In some cases, the gastric skiagram fails to show any abnormality, despite strong clinical evidence of ulcer; but in these instances a bismuth shadow remains after the stomach is empty. It represents a notch in the posterior wall, which did not succeed in demonstrating itself on the contour of the gastric shadow.

But if this appearance also fails as an indication of an ulcer in the fundus of the stomach, it must be looked for in the pylorus or duodenum. A pyloric ulcer is only visible in a skiagram if it has led to pyloric spasm or cicatricial stenosis. In either case there are the clinical symptoms of retention, and the skiagram shows a food residue after six to twenty-four hours. The part played by spasm in the stenosis can be determined by giving atropine ($\frac{1}{60}$ gr. hypodermically) or papaverin ($\frac{3}{4}$ gr. hypodermically or by the mouth) which may overcome the spasm. Perhaps a more certain differentiation can be arrived at by noting the time taken for the expulsion of water from the stomach (Holzknecht).

If the narrowing of the pylorus is merely due to spasm, 200 grms. of water pass through in an average time of sixty minutes; if the stenosis is organic, twice this time is required. The determination is made by putting in the water two bismuth pills, one of which floats while the other sinks. When both pills have reached the same level, it is obvious that all the water has left the stomach. It is important to distinguish between spastic and organic stricture, because the former often recovers of itself, whilst the latter demands operative treatment. If the six-hour residue consists of the greater portion of the bismuth meal, indicating that it would take at least twenty-four hours for the complete emptying of the stomach, this last test becomes quite superfluous, because such a degree of stenosis always signifies an organic cause.

Pyloric ulcers never give rise to the formation of notches on the skiagram.

But if a pyloric ulcer is incapable of being demonstrated, despite the persistence of its clinical signs, we are compelled, by exclusion, to suspect a **duodenal ulcer**; i.e., an ulcer near the pylorus, but definitely on its distal side. The chief skiagraphic indication of such an ulcer is the rapid emptying of the stomach—often within two hours; but this is also the case with disease of the gall-bladder and pancreas. In some cases the emptying of the stomach proceeds rapidly up to a point, and then the process slows down so that a small six-hour residue can be detected, which is due to a supplementary pyloric spasm. The duodenum itself, which never becomes completely filled in the normal state, often presents no change; but if adhesions exist, the shadow of the first segment when full, will appear to be irregularly limited, or distorted. At other times a so-called duodenal stain can be detected in the duodenum after the stomach is entirely empty. This, of course, indicates the remains of a little of the bismuth meal. This duodenal stain (fig. 183, *i*) is however, not confined to duodenal ulcers; it may be present whenever the shape of the duodenum is altered and pockets are formed by distorting scars.

It is clear from the foregoing, that the diagnosis of ulcers of the body of the stomach and of the pylorus depends essentially upon positive signs; whereas the diagnosis of duodenal ulcer is in most cases the result of a process of exclusion. This explains the relative uncertainty of the latter diagnosis.

An indirect aid to the recognition of changes in the pylorus, situated low down, or of changes in the duodenum, is afforded by the degree of mobility of these structures in different postures of the body. Our observations show that when the stomach contains 400 c.c. of bismuth meal, the pylorus moves up with a slight median inclination to the extent of 7 to 10 cm. (the level of 2 to 2½ vertebræ), when the patient changes from the recumbent to the erect posture. This mobility is scarcely affected at all by a benign ulcer, but is considerably diminished in malignant disease, the degree of mobility being limited to the extent of one vertebra only.

(2) THE DIAGNOSIS OF SECONDARY INNOCENT CHANGES.

A skiagram may reveal two sequelæ of ulcers. It may show that an ulcer has perforated the stomach wall through Haudek's notch, or that stenosis has occurred in the middle of the viscus, i.e., the formation of an hour-glass stomach. Pyloric stenosis is indicated by dilatation of the stomach and retention, and the degree of stenosis is shown by the shape of the gastric circumference, because the narrower the ulcer the more the stomach dilates in its broad diameter. At the same time the stomach sinks lower, and the process of emptying becomes slower. In duodenal stenosis the retention is very rare. The thin duodenal contents, mixed with bile and pancreatic juice, flow more rapidly through the constricted duodenal area than they flow through a pyloric stenosis before they are thinned out.

The *hypertrophic pyloric stenosis of infants* should be mentioned in this connection. It is a condition which may lead to the most intense degree of malnutrition, but it is not at all clear whether it is due to genuine hypertrophy of the pylorus or to its mere spasm—probably both factors are concerned; but cases occur which demand surgical treatment, whatever be the cause.

(3) THE DIAGNOSIS OF CANCEROUS DEGENERATION OF AN ULCER.

General pathology indicates that a gastric ulcer is susceptible of undergoing cancerous degeneration, and clinical experience establishes this as a fact, especially in regard to pyloric ulcer. The frequency of this change has, however, been greatly exaggerated. Available statistics show that hardly 2 per cent. of ulcers, diagnosed clinically, undergo this degeneration, and obviously if undiagnosed ulcers are included, the percentage would be less. This malignant conversion is suggested, if the symptoms of an old-standing ulcer become rapidly worse, especially in the direction of stenosis, if hyperacidity gives way to deficiency or absence of hydrochloric acid, and if a little blood is constantly present in the stools. Obviously the matter is no longer in doubt, if there are unequivocal signs of cancer of the stomach, such as secondary deposits, in the abdomen, the presence of ascites, increasing emaciation, and anæmia, even without definite stenosis or severe hæmorrhages.

The skiagram is also suggestive of this change if we find that a Haudek's notch is gradually giving way to a loss of shadow, and if the termination of the pylorus which usually appears quite smooth when the scar of an ulcer is present is replaced by an irregular cancerous outline. The comparative fixation of the pylorus, *i.e.*, its slight mobility on the change from a recumbent to an erect posture, is also of significance. If this mobility does not extend beyond the range of a half to a whole vertebra, the probability of cancer is very great, as already stated. Nevertheless, the etiological connection between an antecedent ulcer and a present cancer cannot always be taken for granted, because the two diseases may arise quite independently.

(4) PRIMARY CANCER OF THE STOMACH AND DUODENUM.

We have already discussed in a general way how we arrive at the probable diagnosis of cancer; we shall now study the symptoms in more detail, and endeavour to ascertain whether we can determine the variety of cancer with any accuracy.

A decade or so back there was no need to hurry with the diagnosis of cancer, because in any case the patient was doomed; to-day, however, the cure of the condition depends upon early diagnosis. Unfortunately large numbers of cases proceed along their fatal course without giving rise to any symptoms until it is too late for operation, but very many cases still remain where any early diagnosis could have been made if the medical attendant had thought of carcinoma, and submitted the patient to a systematic examination, instead of dosing him with drugs for indigestion.

The first thing which ought to be looked for instinctively because it is the most palpable sign is *tumour*. For this purpose the examiner should sit by the side of the patient, who must lie horizontally with abdomen relaxed, and palpate with slight pressure along both sides of the vertebral column from above downwards. If the abdominal integuments are soft, it is possible to feel in this way the greater curvature, and sometimes also the pylorus. If there is much displacement of the stomach, the lesser curvature may also be palpable. A little below the transverse colon can be felt, unless it has become displaced farther down. An indurated pylorus usually feels like a hard movable pad, lying transversely, which slips away from the palpating finger with a sudden jerk—just as the normal pylorus. An indurated pylorus may be confused with a pathologically thickened pancreas (chronic pancreatitis or cancer), and even with a normal pancreas, in very thin patients. The pancreas can sometimes be palpated in its entire extent in this type of patient,

Most cancers of the stomach grow from the lesser curvature and if they are at all palpable appear to be quite large. But they are often hidden under the liver and therefore cannot be felt, even if they have grown to considerable size.

We do not always find the growth where we expect to find the normal stomach. If the cancer occurs in a displaced stomach, or if the stomach sinks because of the weight of the growth, the tumour is usually found in the left half of the abdomen, or even in the left pelvic cavity. The connection of the tumour with the stomach is indicated by the fact, that it can be moved round the segment of a circle, whose centre roughly corresponds with the normal position of the pylorus. That the tumour is not connected with the colon is proved by the fact that both the transverse and the descending colon can be palpated separately from it.

If palpation is indecisive, despite a well founded suspicion of disease, we must proceed to examine the patient under an anæsthetic or to take a skiagram.

No significance is to be attached to a negative result of palpation. On the other hand, as we have already stated in discussing gastric ulcer, if something is found on palpation it does not necessarily follow that it is cancer, for tumour-like indurations of the gastric wall and especially of the pylorus occur in cases of ulcer (callous ulcer). In some cases the associated symptoms will establish the correct diagnosis; but in others, the diagnosis cannot be made until the abdomen is opened, and perhaps even then the aid of the microscope may be required. It follows therefore that every tumour-like structure must be treated as if it indicated cancer.

So far as the chemistry of the stomach is concerned, there is nothing to add to what has been stated previously.

Many attempts have been made by examining the gastric contents —especially the proportion of albumin in the water used for irrigating the stomach—to render the diagnosis more certain (Salomon). No method has however proved sufficiently reliable, to warrant general employment.

The position is not much better in regard to the various *serum tests* which have been put to exhaustive trials in cases of cancer of the stomach. The examination of the blood for Anti-trypsin, and the Meiostagmin reaction certainly give frequent positive results in cancer, but the complexity of these tests is greater than the reliability of their results. For the moment it seems doubtful whether the application of Abderhalden's principles to the diagnosis of cancer, will fare any better.

We now proceed to deal with the separate varieties of cancer of the stomach.

(1) CANCER OF THE CARDIAC END OF THE STOMACH.

This soon leads to symptoms of stenosis, and the clinical picture resembles cancer of the lower end of the œsophagus. This type of cancer can be readily seen through the œsophagoscope. If it proliferates towards the stomach, it may be detected in a skiagram taken with the patient in the Trendelenburg position. In these cases, pain radiating towards the back, is an early symptom.

(2) CANCER OF THE BODY OF THE STOMACH.

As the growth is not at one of the orifices of the stomach, it is characterized by an absence of symptoms, which may not come on, until the case is beyond operation. It usually starts on the lesser curvature and infiltrates the anterior and posterior surfaces of the stomach, saddlewise. The larger portion of the cancer is tumorous in nature, but there is deep destruction in its centre which is seen in a skiagram as irregularly circumscribed areas of loss of shadow. These areas can usually be distinguished with ease from similar areas caused by swellings lying against the stomach.

These carcinomata inevitably lead to the development of stenosis, although it may not come on until the later stages. As a rule the stomach empties itself with abnormal rapidity, the skiagram showing the entire bismuth meal in the intestine within 2—4 hours.

The flat ulcerative form of cancer of the body of the stomach, is a somewhat rare variety. It corresponds to rodent ulcer of the skin. In these cases there is an obliteration of the stomach shadow owing to the rigid apposition of its walls. This form is closely allied to the cancerous infiltration without any extensive ulceration, which is known as Brinton's cirrhosis—the linitis plastica of the French—in which condition the stomach is converted into a rigid tube of small capacity, i.e., the flask-shaped stomach. The skiagram shows a narrowing of the gastric shadow, with a funnel-shaped expansion towards the cardia because the upper portion of the stomach is the last to be involved. If stenosis developes eventually, regurgitation of food occurs, just as happens in cases of cancer of the cardiac end of the stomach, but in contrast to this latter the stomach tube can be easily passed, though the stomach cannot be inflated through it, or filled with fluid to any considerable extent.

The polypoid variety of cancer of the fundus of the stomach is extremely rare. It is so soft that it can hardly ever be felt externally, even after the abdomen is opened. This variety bleeds very readily so that the patients look like cases of pernicious anæmia. All cases of intense anæmia, without obvious cause, ought to be submitted to an exhaustive gastric examination, and a skiagram should be taken. In one of our cases the diagnosis was made by examining a piece of tissue brought away by the stomach tube. There may be several of these polypoid growths present in the stomach, just as in the intestine.

(3) CANCER OF THE PYLORUS.

Cancer of the pylorus is not only palpable earlier than cancer of the lesser curvature, but its clinical signs also appear earlier, because it so soon produces a stenosis of the pyloric orifice. It can also be diagnosed at an earlier stage than cancer of the body of the stomach.

In early cases, the skiagram exhibits an obliteration of the pyloric area, and a certain amount of delay in the passage of food. At the same time the pylorus contracts adhesions, so that it is not drawn over to the right when the stomach is full, as occurs in cases of ulcerative stenosis. In the latter, there is a tendency for the pylorus to be dragged some distance over to the right, whereas in cancerous stenosis the whole of the filled portion of the stomach lies in the left half of the abdomen. If the cancer has developed slowly and no adhesions have occurred until a late stage, the skiagram will exhibit the same type of dilatation as occurs in ulcerative stenosis, and the only suggestion of cancer will be derived from the irregular limitation of the stomach shadow towards the pylorus. But in the absence of this sign, not even the skiagram will differentiate carcinoma from innocent stenosis.

Sometimes the growth is so circumscribed that no definite loss of shadow is demonstrable. At this stage, there may be no dilatation, and the stomach may empty itself within six hours. The only indication of a commencing stenosis may be shown by an abnormal peristalsis (several deep waves on the same skiagram). The presence of a conical process of shadow in the obliterated portion, is highly significant—for it shows the track of the bismuth meal amid the masses of growth.

It happens sometimes, especially if the skiagram is taken in the erect posture, that some bismuth-free gastric juice forms a layer over the bismuth emulsion, and interferes with the shadow. This must not be mistaken for an obliteration of the pylorus, due to cancer.

CHAPTER XLI.

ACUTE PERFORATION OF GASTRIC AND DUODENAL ULCERS.

THE sudden perforation of an ulcer into the abdominal cavity is no longer a matter of mere gastric or duodenal disease, because the rapid onset of peritonitis will dominate the symptoms and command the entire clinical interest. There is no other form of peritonitis which demands such rapid comprehension of the situation, because most cases operated on within 12 hours recover, while of those operated on after 24 hours, only one fourth survive.

If a person—remarkably enough it generally happens in a man—suddenly experiences severe pain like the thrust of a dagger in the epigastrium, followed by rigidity of the abdominal muscles, local pain on pressure, acceleration of pulse and occasionally a rise in temperature, we should think of a perforated ulcer, although the patient may not have manifested any signs of gastric ulcer previously. The perforation is often, but not always, associated with collapse, from which the patient may temporarily recover before the classical symptoms of peritonitis set in. In such cases, even the delay of half a day "for the purpose of better observation," means to abandon the patient to almost certain death. If we first see the case at a later stage there is either general peritonitis, the origin of which cannot be traced, or there may be the condition termed by Lennander peripheral peritonitis. After perforation the gastric contents flow towards the anterior surface, preferably under the liver towards the right lumbar region, thence into the true pelvis, and the peritonitis eventually ascends along the left side, without always affecting the convolutions of the small intestine, which are covered by omentum. This peripheral form remains longer accessible to surgical treatment than the central form. If we find the patient, as is usually the case on the second or third day, in a condition of cyanosis with a tympanitic abdomen, cold extremities, and a thread-like pulse, it is always questionable whether operation is worth while. It may only shorten the patient's life by a few hours and redound but little to the credit of our art.

We now deal briefly with some details. The *age* of the patient is of little diagnostic aid, though most perforations occur between 20-50 years. I have however come across cases in a girl of 13 years, and in persons beyond 70. Sometimes the *previous history* clearly indicates or suggests the possibility of a gastric ulcer, but this is not by any means invariable. Sometimes, the perforation is preceded by premonitory symptoms of peri-gastritis, but these symptoms are usually misunderstood. There is however no possibility of misunderstanding the absolute suddenness and the intense violence of the pain of perforation. The patient is always able to distinguish it from his accustomed gastralgia, or from any premonitory pain. There is no other acute abdominal disease which renders even the strongest man so completely and unexpectedly hors de combat, forcing him to lie absolutely still, breathing spasmodically and groaning.

The initial pain is never absent, but it may be less severe than usual if the perforation does not exceed a pin point in size as sometimes happens. The pain often radiates into the back especially between the scapulæ, or into the left shoulder and arm, just as the pain of biliary disease radiates into the right shoulder. If the pain is situated towards the right, and the rest of the clinical picture is typical, it points to perforation of a *duodenal ulcer*, which otherwise resembles perforation of a gastric ulcer, in all respects.

If our diagnosis be attended by any doubt, it is converted into a certainty, in cases wherein the patient has asked for an alcoholic stimulant immediately after the onset of the pain. It is remarkable that patients do not ask for alcohol in most other acute inflammatory diseases of the abdomen, such as appendicitis, or cholecystitis. The feeling for a stimulant only occurs in perforation, because of the suddeness of the pain and the intense prostration.

The localization of the pain in the subsequent course of the disease has less significance for diagnosis, because this depends upon the direction taken by the stomach contents. The pain usually localizes to the lower abdomen or the right side, less often the left side.

This explains why perforation of the stomach has often been mistaken for disease of female pelvic organs.

Pulse, *temperature* and *respiration* behave as in any other perforation of a viscus. The pulse is slightly accelerated, but may remain so full, strong, and quiet for the first few hours, probably through vagus stimulation, that the inexperienced may entirely dispose of the idea of perforation. A normal temperature does not exclude perforation, but, on the other hand, a rise in temperature shows that the case is not an ordinary stomach ache. Respiration is hurried, shallow and thoracic. It is an error to suppose that early vomiting does not occur; on the contrary it is frequently present.

The most important *local signs* are:—

(1) Extensive reflex rigidity, especially in the upper abdominal region, but generally over the whole abdomen, both sides being equally involved.

The degree and extent of the reflex rigidity distinguish acute perforation of the stomach from all other conditions which enter into the differential diagnosis. Those who devote their attention to this sign will not usually find any difficulty in the diagnosis.

(2) The presence of gas in the free abdominal cavity. The remarks already made in connection with laceration of the intestine apply here also: liver dulness is usually present, and there is no escape of gas in most cases; but if gas is present, it may limit itself to a movable collection at the uppermost region of the abdomen, revealing itself by a localized metallic or very tympanitic note.

(3) Friction murmur, and soft friction appreciable by the hand over the gastric area. This sign is rare, but if present is of great value.

(4) Rapidly increasing dulness in the flanks, especially on the right.

Important as it is to diagnose a perforated gastric ulcer early, nevertheless we must not make this diagnosis just because the patient happens to be a girl at an age when gastric ulcer is common. We may easily be misled by *acute intoxications*, especially as a reliable history is not often obtainable in these cases.

Symptoms of a perforated gastric ulcer in a pregnant girl always suggest poisoning—a circumstance not exactly creditable to the "stronger sex."

There is hardly any disease of the abdomen which has not been confused with perforated gastric ulcer, especially *cholecystitis*, *pancreatitis*, *rupture of a pregnant tube*, *torsion of an ovarian tumour*, and *acute obstruction*. Even acute pneumothorax has been taken for a gastric perforation. The same applies to the *crises of tabes*.

It does not take very long to examine the reflexes, and the physician has a just cause for criticism if the abdomen of a tabetic is opened on four occasions for gastric crises, as was recently reported.

The occurrence of signs of perforation in a patient who has undergone gastro-enterostomy usually indicates rupture of a peptic ulcer of the jejunum—one of the darkest spots in the whole of gastric surgery. This risk is greatest after anterior anastomosis.

In some cases the symptoms of perforation come in with less severity, and its signs are limited to the upper part of the abdomen, the lower portion being but slightly rigid or even quite soft. Recovery may take place without operation—but often at the expense of a perigastric or subphrenic abscess. The final result is often the formation of a diverticulum. This type of case occurs, if the perforation is small and the stomach empty, and recent adhesions have formed. Schnitzler has termed this attenuated clinical picture, "a concealed perforation." It is quite impossible to differentiate such cases from mild attacks of acute pancreatitis, if they occur in fat subjects of an age when pancreatitis is prevalent.

CHAPTER XLII.

SURGICAL DISEASES OF THE BILIARY PASSAGES.

NOTWITHSTANDING the better appreciation of the nature of gall-stone disease, which we have gained during the last twenty years, there still remain some practitioners in whose minds the ideas of "jaundice" and "gall-stones" are indissolubly connected. We still hear the assertion "there are no gall-stones because the patient has no jaundice." Jaundice is a symptom which *may* occur in gall-stone disease, but is *not essential* thereto, and is moreover present in many other diseases.

If a patient informs us that his liver is affected, or if we have the impression that he is so suffering, our first thoughts should be of the "medical" diseases—simple *biliary catarrh*, the various forms of *hepatic cirrhosis*, and *acute yellow atrophy of the liver*.

If a patient with indigestion but without pain, becomes yellow but otherwise remains well, we must diagnose *catarrhal jaundice*. But if the general condition is profoundly affected, and the temperature is high, the disease is *infective jaundice*, which may also occur in the epidemic form. This also constitutes the rare malady known as Weil's disease. If a high degree of jaundice is associated with symptoms of severe general illness and rapid loss of strength, we should assume either *acute yellow atrophy of the liver,* as a result of poisoning (phosphorus, arseniuretted hydrogen) or some form of septic infection. A certain amount of jaundice, without any profound anatomical changes in the liver, often exists in acute septic conditions, especially in septic peritonitis, and all experienced observers are acquainted with the terrible dirty greenish look—due to the yellow of the jaundice combined with the blue of the cyanosis—of patients who have succumbed to this disease. A patient who gets about for years, with more or less jaundice, but without any particular pain, but who complains occasionally of slight feverish symptoms and a general deterioration of his health, must be suspected of suffering from *hypertrophic cirrhosis of the liver*—which is merely a chronic infection of the organ with acute exacerbations.

The jaundice of Banti's disease should be mentioned here, in which condition the enlargement of the liver is associated with great splenic hypertrophy. The striking improvement which often follows removal of the spleen in these cases, is tending to bring this etiologically obscure disease, within the scope of surgery. Secondary and tertiary syphilis may also produce jaundice.

The above comprise all the usual conditions which may suggest forms of gall-stone disease. But if the patient's "liver trouble" does not appear to fit in any of these categories, we are justified in concluding, by exclusion, that the case is probably one for surgical treatment. We should accordingly think of gall-stones, malignant growth, abscess of the liver and hydatid cyst.

We may now apply our diagnostic reflections to a few concrete cases.

(1) GALL-STONE COLIC.

In one group of cases, pain is the predominant feature. The patient is seized, at rare or frequent intervals, with severe pain in the upper part of the abdomen or more definitely in the gall-bladder region. Nothing but morphia suffices to relieve the pain which, however, does not last more than a few hours, or at most, a day. There is usually no rise in temperature, nor is there, as a rule, any jaundice, so that the patient, and often the doctor, diagnoses "colic." But gastric colic is really very different to gall-stone colic, both in its onset and in its nature. Pains arising in the stomach generally radiate to the left and towards the back; biliary colic radiates towards the right, even as far as the right shoulder. If gastric pain is caused by an ulcer, it is increased on taking solid food. If caused by hyperacidity, food relieves the pain. In both cases the pain comes on almost regularly at definite hours of the day or night. The pain in an acute attack of gall-stones, so called

biliary colic, is quite independent of food and it occurs at irregular intervals of months or years. This also differentiates it from the pain of duodenal ulcer, which is situated on the right side and comes on at the completion of gastric digestion. This is termed "hunger pain."

A great deal of difficulty is occasioned by the less severe, temporary or persistent pains which precede or follow a severe attack, or by conditions wherein a pronounced attack of acute biliary colic does not occur at all. A stone may be present in the common duct and yet the only symptoms may be those of indigestion—of a reflex character, or due to transient biliary congestion. From this condition, it is only one step to the very numerous class of cases wherein gall stones exist, with a total absence of all symptoms. Thus arises the impossibility of making a diagnosis in many cases, unless deep pressure over the gall bladder, at any rate occasionally, excites the characteristic pain.

We may now return to the attack of colic itself. This may be mistaken for any acute painful seizure in the upper abdomen, viz., for pain caused by *hernia of the epigastrium* or *umbilicus*, renal colic, and the severe conditions to be described in the following section.

Biliary colic may be distinguished from renal colic, even in the cases where there is no palpable swelling to assist in localization, by the position of the reflex muscular rigidity. If we press simultaneously in front of and behind the painful region, in a case of renal colic, the lumbar muscles will become rigid, whereas in a case of biliary colic, it is the right rectus which becomes rigid. It has been stated that in gall-stone disease there is a particularly characteristic painful spot close to the right side of the spine below the twelfth rib; but one must use this diagnostic point with discrimination.

In order to explain the short attacks of biliary colic on an *anatomical basis*, we must assume that they depend either on a mechanical obstruction of the ducts—which used to be considered the exclusive cause—or on spasm of the muscular wall embracing the stone, a condition often overlooked, or on a mild degree of inflammation around the stone incarcerated in the cystic or common duct—an inflammation which runs its course rapidly after reaching its height within a few hours. Unless recurrences are very frequent, surgical aid is unnecessary. We will discuss the *position* of the stone in connection with other forms of cholelithiasis; but it is usually very difficult to localize it in mild attacks. The discovery of the stone in the stools after the attack often shows that the pains are due to the extrusion of the calculus. Some patients are able to exhibit a stone for each attack. There is no jaundice in these slight attacks because the obstruction of the common duct is too brief.

The attacks of pain caused by adjacent adhesions are sometimes indistinguishable from mild biliary colic. These attacks may occur whether the stone has passed naturally or has been removed by the surgeon.

It is often important to be able to decide whether attacks of pain of which a patient has complained were really due to gall-stones. Two practical rules are applicable in this connection. The patient is able to go to his doctor for very many attacks of pain, but the doctor is obliged to come to the patient for biliary colic. An anodyne prescription suffices for ordinary pain, but a hypodermic injection of morphia is indispensable in the pain of gall-stones. Exceptions are rare.

(2) ACUTE CHOLECYSTITIS.

The following case raises somewhat different problems of differential diagnosis, in regard to acute biliary colic.

A patient is suddenly seized with symptoms of pyrexia and severe pain in the right side of the abdomen. He vomits on one or more occasions, indicating peritoneal irritation. The colour of the skin is normal, the pulse is good, the abdominal muscles, especially on the right side, are tense or become so, as soon as they are touched, and we think of appendicitis, because of its frequency or perhaps because it is in fashion. On careful examination, it will, however, be found that the centre of the painful area and the rigid musculature is not situated in the line joining the anterior superior spine and the umbilicus, or below it, as is the rule in appendicitis, but higher up in the region of the gall-bladder. Percussion probably shows that the liver dulness projects below its normal limits, even extending as far as the level of the umbilicus. If palpation is possible, despite the muscular rigidity, an area of resistance, with its lower border circular in shape will be felt connected with the liver. If this be the physical condition found, there can be no doubt that the case is one of *acute cholecystitis*, and if the pyrexia persists for many days it is certainly of a suppurative character. The position of the pain and the pyrexia often suffice for the diagnosis, without it being possible to demonstrate the typical lobular or tongue-shaped area of resistance. In such case we may assume that a contracted gall-bladder, concealed under the liver has become inflamed, and we will generally obtain an old history of gall-stones, in confirmation of this assumption.

A recent perforation of a *gastric or duodenal ulcer* might suggest acute cholecystitis. But in perforation, the reflex muscular rigidity very quickly affects the whole abdomen, and generalized peritonitis soon sets in if the case is neglected, symptoms which only occur in cholecystitis if an infected gall-bladder perforates into the free abdominal cavity.

Pancreatitis and *pancreatic hæmorrhage* should also be mentioned in this connection, but in these, the pain is rather to the left, and the pulse soon becomes thready and rapid.

If the gall-bladder and appendix were invariably in their normal positions, differential diagnosis would be very easy. This is, however, not the case. Sometimes the gall-bladder reaches as far as the ileo-cæcal region, whether the liver be movable or not. But much more frequently, the appendix is high and directed outwards. At other times its position is in close proximity to the gall-bladder especially when there is a "mesenterium commune ileo-cæcale." I once saw it, at an early operation, strung up by a lateral band of connective tissue, close to the gall-bladder behind the liver.

These abnormalities in the position of gall-bladder and appendix have led to many errors in diagnosis. The following is an instance :—

A female, aged 40, was operated on by an experienced surgeon for a "perityphlitic abscess," and she subsequently had a persistent fistula in the ileo-cæcal region. Two years later she was admitted to hospital suffering from acute hemiplegia. She died in a few days, and the autopsy showed that the fistula, which opened at the upper part of the inguinal region, led into the gall-bladder, which contained a large stone. The cystic duct was closed. The hemiplegia was the result of a cerebral abscess, in which were found diplococci similar to those in the gall-bladder. Neither the surgeon nor the physician was fortunate in the diagnosis of this case, although they were both experienced observers.

When the inflammatory process occurs above the line joining the anterior superior spine to the umbilicus, we should think of appendicitis if the pain or resistance reach far *towards the side*, and if the lumbar muscles respond to pressure, by contraction ; but any inflammatory process internal to the external border of the rectus must be ascribed to the gall-bladder. If jaundice supervenes in addition, the beginner will immediately decide in favour of gall-stones. This is usually a good guess, because the extension of the inflammatory swelling from the gall-bladder to the common duct often leads to a mild temporary jaundice. But, on the other hand, we often find a certain amount of jaundice in appendicitis, not necessarily in the severest cases. If the appendix lies near the gall-bladder, so that the biliary passages are secondarily involved in the inflammation, the jaundice may become very pronounced.

A young man was brought into the hospital with a high temperature, severe jaundice, and abdominal resistance reaching outwards from the lateral border of the rectus immediately adjoining the liver, and extending as far as the crest of the ilium. Despite the jaundice a diagnosis of appendicitis was made, based upon the lateral position of the resistance. This was confirmed at the operation.

A rule to the following effect often enables a correct diagnosis to be made, when the painful spot is situated just on the border line: if the dulness reaches as far as the flank, the case is appendicitis, but if an intestinal note can be elicited externally to the painful area, cholecystitis is present.

If we still remain in doubt after taking all physical signs into consideration, we must be guided by the rule that appendicitis is more probable the younger the patient, and gall-stones the older he is. Nevertheless I have twice seen girls, aged 18, sent in for appendicitis, but who were really suffering from cholecystitis.

The possibility of a *renal disease* should also be borne in mind. The following case illustrates the difficulties of diagnosis.

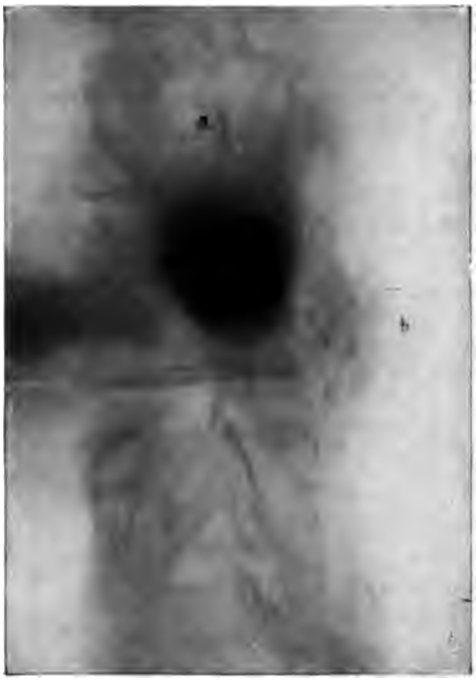


Fig. 185.—*a*, Bismuth shadow in duodenum; *b*, calcium carbonate gall-stones. See also fig. 187 (5).

A woman, aged 35, was subject to periodical attacks of pain in the upper abdomen, and these were attributed to gall-stones. Indeed a very adherent gall-bladder, full of stones, was removed, but the attacks recurred. They were of short duration, associated with some rise of temperature, and the pain was situated to the right of the umbilicus. Appendicitis was diagnosed. I saw the patient at the end of an attack, but there was no tenderness on pressure, either over the gall-bladder, the appendix or the kidneys. There was pus in the urine coming from the right kidney. Examination of the biliary passages and of the appendix showed nothing abnormal, but the kidneys were situated low down, fixed and somewhat enlarged. All the skiagrams taken showed an indefinite shadow of a calculus. Despite the gall-bladder full of stones, the renal calculus was the real trouble—already at the first operation. The malady was ultimately due to gonorrhœal cystitis with ascending infection.

A special form in which cholecystitis may manifest itself should be referred to. Just as cases of gall-bladder inflammation come to the surgeon with the diagnosis of appendicitis, so do others present themselves which have been diagnosed as "ileus." Indeed cholecystitis may at its beginning or during its progress, cause adhesions between the intestine and gall-bladder, kinking or inflammatory infiltration of the intestinal wall, extensive, serous or sero-purulent peritonitis, which may either produce mechanical obstruction, or the toxic symptoms of an obstruction. A correct diagnosis can only be made from the previous history of the patient, and the presence of pain and resistance, or at least of a localized area of pain on pressure, in the region of the gall-bladder.

(3) GANGRENOUS CHOLECYSTITIS.

In the forms of gall-bladder inflammation so far described, the disease has been limited to the gall-bladder and its immediate vicinity. But if the clinical aspect is much more severe and septic symptoms develop rapidly, or if an increased resistance follows upon a very acute stage, we should suspect *gangrenous inflammation* of the gall-bladder, with extensive participation of the peritoneum.

FIG. 186.—Skiagram of two encrusted gall-stones. (Bismuth meal in stomach.)

A man, aged 65, was suffering from severe inflammatory symptoms in the upper abdominal region. At first they seemed to abate, but subsequently a diffuse resistance developed in the gall-bladder area, with persistent high fever. Operation was performed at this stage, and a great cavity filled with offensive pus was encountered, bounded by the liver, stomach, duodenum and large intestine. A shreddy dark grey membrane, which still preserved the shape of the gall-bladder, was hanging in the cavity, but it was completely necrotic and contained a gall-stone.

As in the other form, *jaundice* is also here merely an accessory symptom, which is not essential to the clinical picture of cholecystitis. But if it is present—and it is usually transitory—we must infer that a phlegmonous inflammation has invaded the biliary passages from the gall-bladder and has temporarily arrested the free flow of the bile.

We have hardly referred to the gall-stones themselves. It is not the gall-stones which we diagnose, but the inflammatory changes

(1) Bilirubin-lime calculi from the intra-hepatic biliary ducts, containing neither calcium carbonate nor calcium phosphate.

(2) Gall-stones mainly consisting of cholesterin, with bilirubin in the centre.

(3) Facetted gall-stones, consisting of cholesterin, pigment and some calcium phosphate.

(4) Mammilated gall-stone with a deposit of Ca. and Mg. carbonates and phosphates.

(5) Gall-bladder with calculi resembling thorn-apples, consisting almost exclusively of calcium carbonate (from case shown in fig. 185).

(6) Ureteral stones containing organic material, carbonate and phosphate of calcium and magnesium.

(7) Vesical stone, consisting of calcium oxalate.

(8) Urethral stone, containing organic material, calcium and magnesium phosphate.

(9) Salivary stones, containing organic material, calcium phosphate and a little magnesium phosphate.

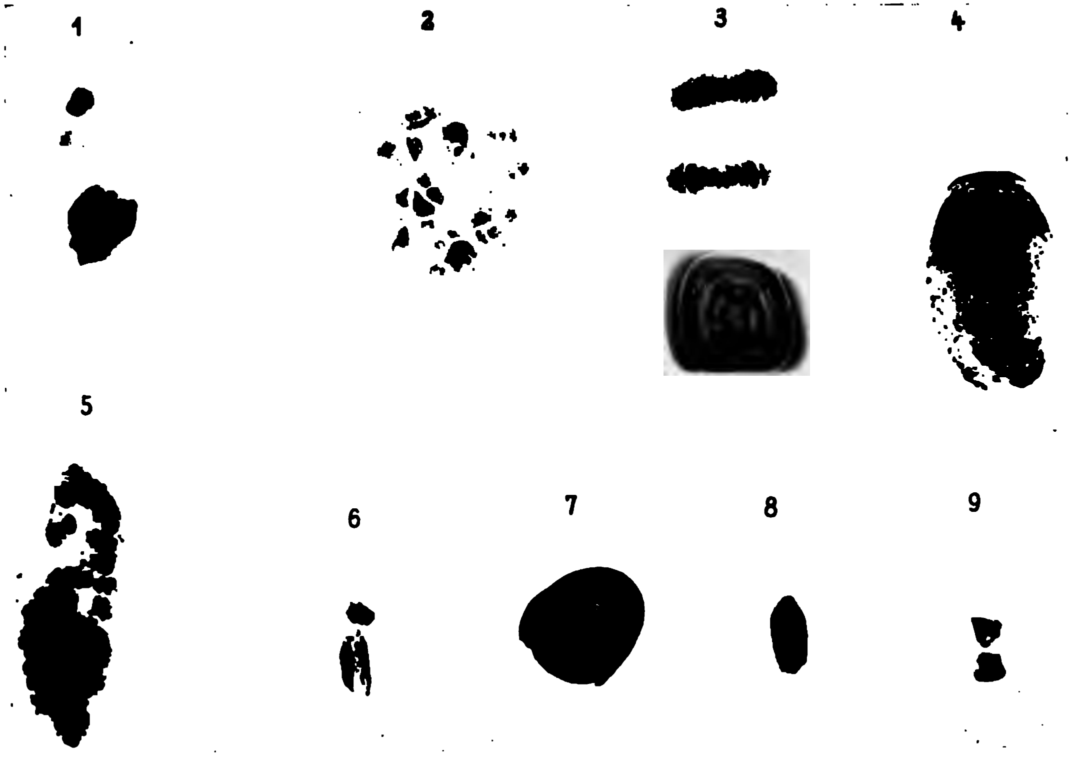


FIG. 187.—Comparative illustrations of skiagrams of a few calculi.

which their presence causes. We know very well that slight catarrhal inflammation of the biliary passages often occurs after gastric and intestinal catarrh, and that very acute cholecystitis may supervene during the progress of typhoid fever, dysentery and cholera, without any formation of stones. But these only account for one-tenth of the cases of cholecystitis which come under observation. It follows, therefore, that a patient who suffers from cholecystitis without any of the antecedent illnesses just mentioned, most probably is the subject of gall-stones. But it is very regrettable that a purely academic discussion on the matter should be responsible for the delay of a timely operation, and that a stone, whose existence may be doubtful, should eventually assert itself by gall-stone obstruction and fatal intestinal perforation, examples of which I have witnessed.

It is a matter of secondary consideration whether the stone is in the gall-bladder or in the cystic duct. In the latter case there will be more acute exacerbations of jaundice than if it is situated in the gall-bladder.

Many attempts have been made to enlist the service of skiagraphy in the diagnosis of gall-stones. But as their main constituent is cholesterin, a substance which is almost equally transparent to the X-rays as the soft tissues of the human body, not much is to be anticipated from this method of examination. Positive results have only been obtained in the very rare cases of pure calcium carbonate stones, and with cholesterin stones which contain a large amount of calcium and magnesium salts (or are encrusted therewith) as a result of secondary infection. Pigmented lime stones, on the other hand, are so loosely built up that they cannot be detected in a skiagram. The accompanying figure (187) shows how a few of the most important calculi are brought out on a skiagram, and figs. 185 and 186 represent calcium carbonate stones revealed by skiagraphy of the living body (found accidentally after a bismuth meal).

(4) OBSTRUCTION OF THE COMMON BILE-DUCT.

This presents a totally different clinical picture. Jaundice is the predominant feature; localized dulness and swelling are hardly ever present. The liver is enlarged, the urine contains much bile pigment and the stools are colourless. These symptoms indicate that some obstruction exists in the common duct, and we have to determine whether this is caused *only* by a **stone** or whether a **malignant new growth** may be present. In cases of stone the obstruction is not merely caused by the foreign body, but also, and to a great extent, by the accompanying inflammatory condition, and therefore the biliary content of the stools and the degree of jaundice are liable to variations. But if the obstruction is caused by carcinoma there is a constantly increasing mechanical pressure exerted by the new growth, and therefore the stools are always free from bile and the jaundice is persistent. If, in addition, we realize that the pain is not due to congestion of the bile, but to the inflammatory process, we will understand that attacks of pain point to gall-stones, whereas painless jaundice suggests a tumour. These are two fundamental differences between the two conditions. These remarks concerning the attacks of pain apply equally well to exacerbations of high temperature, with or without rigors. But we must be on our guard against diagnosing gall-stones merely because there is a previous history of biliary colic. In at least five-sixths of the cases of cancer of the gall-bladder there has been a previous history of gall-stones. How, then, are we to make a correct diagnosis ? An example may perhaps indicate this.

A middle-aged female suffered for many years with regular attacks of recurrent gall-stones, in testimony whereof she produced a box full of stones which she had passed spontaneously. The last "attack" was a particularly mild one, but the jaundice, which was more intense than on previous occasions, did not pass off in the usual manner; indeed, it had persisted for several weeks unaltered. The general condition had also suffered much more than in previous attacks. These few indications sufficed to establish the diagnosis of cancer. The slight pain, but the persistent jaundice, were the cardinal symptoms, and the commencing cachexia was corroborative evidence.

The importance of jaundice existing without pain is shown by the following case :—

An aged female suffered from complete obstruction of the common bile-duct, but had no pain. A firm, somewhat nodular resisting mass could be felt in the region of the gall-bladder. Diagnosis : carcinoma. The operation revealed an inflamed gall-bladder filled with pus and stones, but no cancer. The history, however, suggested very strongly that there must be something more present, and as a matter of fact, a deeply seated cancer, which probably originated in the duct, was found.

Physical examination, on the whole, affords little definite information. Palpation is only conclusive if a large irregular nodular tumour is found—but this is exceptional. Nodulated swellings which do not exceed a goose's egg in size, may be the result of gall-stones.

Courvoisier's rule, if carefully applied, is very useful. If we find a tensely filled gall-bladder in a case of chronic obstruction of the common duct, we may conclude that the walls of the gall-bladder are capable of being stretched, and therefore are not chronically inflamed. But as calculus obstruction generally occurs after prolonged suffering from cholelithiasis, that is to say, after the gall-bladder has undergone chronic inflammation and contraction, it follows that a distended gall-bladder contra-indicates calculous obstruction, and suggests the presence of a new growth pressing upon the common duct, but leaving the gall bladder itself free. On the other hand, if the gall-bladder cannot be felt, it is an argument against a tumour, and is in favour of obstruction by a stone. This latter conclusion is, however, only applicable if the tumour does not originate in the gall-bladder itself. Primary cancer of the gall-bladder is very frequently quite unable to be felt, or only as a small nodular swelling connected with an inflamed contracted gall-bladder, just as in the case of calculous obstruction. On the other hand, calculous obstruction sometimes occurs when the gall-bladder is still healthy, or at least still remains capable of distension, and then it can be felt on palpation just like a congested gall-bladder due to obstruction by a growth.

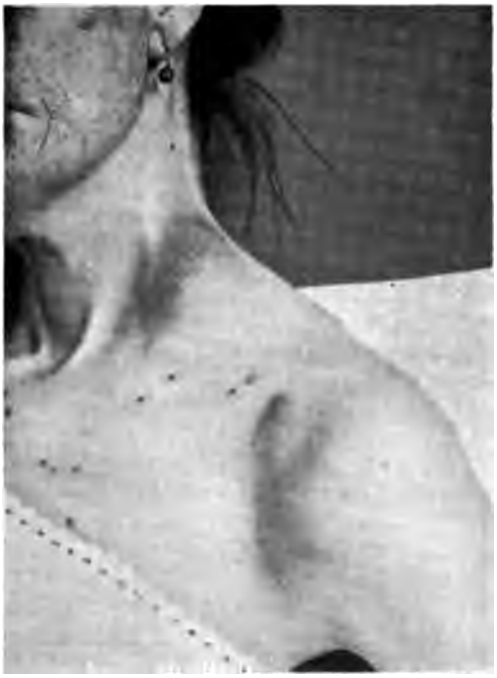


FIG. 188.—Secondary deposits in cervical glands, in case of cancer of gall-bladder.

We must always search for *secondary deposits* (e.g., nodules on the upper surface of the liver, Virchow-Troisier's glands beneath the insertion of the sterno-mastoid), and especially examine the abdomen for a *free effusion*. This symptom may be conclusive, even when nothing else appears to point to cancer.

A strong man, aged about 40, who had no previous history of biliary disease, suffered from such severe pain of the gall-bladder that a large amount of morphia had to be prescribed. A diagnosis of gall-stones was made, and this appeared to be confirmed by the presence of jaundice. But the jaundice persisted, and then a slight fluid effusion came on, as the first positive sign that the case was one of cancer. Œdema of the legs soon came on as confirmatory evidence, and the fatal result was not long delayed.

Ascites occasionally comes on in cases of obstruction by stone, as a result of the secondary cirrhosis of the liver, which is due to the biliary congestion.

If the symptoms gradually and spontaneously vanish, after we have diagnosed *calculous obstruction*, it does not signify that our diagnosis was wrong, nor can we be sure that the stone has passed. As soon as the inflammatory swelling has subsided, the bile can flow freely alongside the stone, which may remain latent in the common duct. If it still remains there, after six to eight weeks, it should be removed by choledochotomy. The spontaneous passage of large stones into the intestine is not usually affected *per vias naturales*, but through a fistulous opening between the gall-bladder and intestine.

If we have arrived at the diagnosis of *obstruction by tumour*, we must determine the situation and origin of the new growth. But this is not often possible. Cancer of the gall-bladder so often runs its course without symptoms until it gains a great depth and obstructs either the common duct or the hepatic duct, causing the same symptoms as the rare primary cancer of the common duct, or cancer of the head of the pancreas. Palpation does not often yield any positive information. Deficient digestion of fats would strongly suggest some change in the head of the pancreas, but it would not differentiate between a new growth and chronic pancreatitis.

(5) HYDROPS OF THE GALL-BLADDER, CHRONIC EMPYEMA.

Cases occur, wherein a single pear-shaped tense swelling which can be felt in the gall-bladder region, indicates disease of the biliary passages. If this swelling is only slightly movable, and is somewhat tender on pressure, and there is also a history of inflammatory attacks the diagnosis of **chronic empyema** of the gall-bladder may be made. But if the swelling is not tender on pressure, if it is strikingly movable, and the history is negative, the diagnosis can only be **hydrops** of the gall-bladder. The cause is usually a small solitary, oval, finely lobulated stone of pure cholestrin, incarcerated in the cystic duct.

A young woman was sent to be operated on for an abdominal tumour. A tensely elastic tumour of the size of a small fist was found in the upper part of the abdomen. It was situated on the right of the spine, but it could easily be pushed to the left, and would remain in the left hypochondrium. On marking out on the surface of the abdomen the circuit of the tumour, the segment of a circle was described, whose mid-point was below the liver. This rendered some connection with the gall-bladder very probable. The patient suffered no pain and had no jaundice, and she had complained neither of her liver nor gall-bladder. Apparently, the swelling was functionally distinct from the biliary system. It was ascertained that the swelling varied in size, but the fluctuations were not associated with any pain. All this pointed to hydrops of the gall-bladder. At the operation the contents were found to consist of a slightly mucoid, limpid fluid, and a solitary stone was discovered in the cystic duct, as anticipated.

Sometimes the clinical picture leaves a doubt in the diagnosis between empyema and hydrops of the gall-bladder. This usually occurs when an empyema has quieted down spontaneously; and if the gall-bladder be punctured at the operation, a clear or very slightly turbid fluid will make its appearance at first, and then a thick purulent deposit will exude.

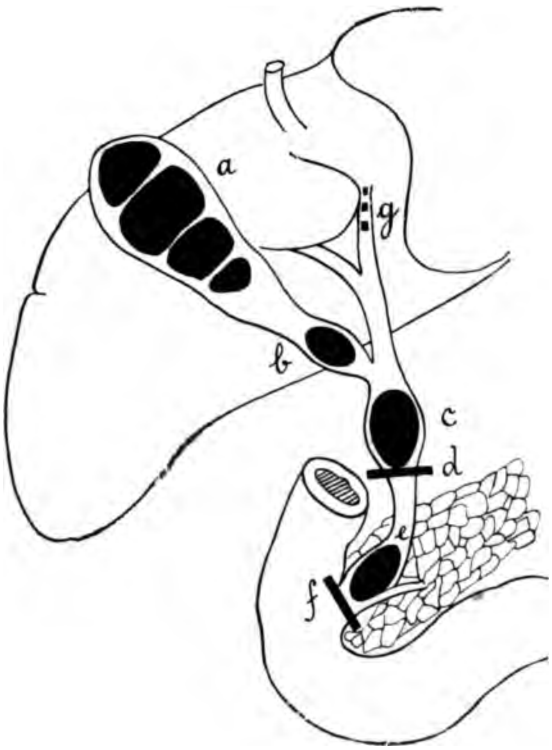


FIG. 189.—General diagrammatic view of the forms of obstruction of the biliary passages.

(*a*) Faceted stones, or large single stone in the gall-bladder. *Without inflammation*, no symptoms. *With inflammation*, cholecystitis, severe spontaneous pain and also on pressure, fevers, rigors occasionally, and vomiting. Usually no jaundice, stools coloured, urine clear. More or less rapid subsidence, or transition to chronic empyema.

(*b*) Solitary stone in cystic duct, oval, generally wedged in firmly. *Without pronounced inflammation*, hydrops of gall-bladder, no jaundice, stools coloured, urine clear, no pain. *With inflammation*, empyema of gall-bladder, symptoms of cholecystitis as in *a*, but gall-bladder larger, and jaundice somewhat more frequent.

(*c*) Stone in upper part of common duct. *Without inflammation*, little or no pain. *With inflammation*, obstruction of common duct of varying degree, jaundice, pruritus, urine brown. Stools yellowish or greyish-white. Liver enlarged, gall-bladder unusually small, pancreatic digestion normal. Frequent attacks of pain, high temperature, rigors. Ascites in the later stages.

(*d*) Obstruction in upper part of common duct, due to a tumour (cancer of gall-bladder, cystic duct or common duct). Persistent severe jaundice, pruritus, urine brown, stools constantly grey. Attacks of pain, high temperature and rigors usually absent. Early ascites. pancreatic digestion normal.

(*e*) Obstruction by stone in lower part of common duct. *Without inflammation*, no pain or indefinite. *With inflammation*, as at *c*, but jaundice more persistent. Occasionally disturbance of pancreatic digestion.

(*f*) Obstruction by tumour at lower part of common duct. (Cancer, more rarely chronic inflammation of head of pancreas, cancer of duodenum), as at *d*, but generally disturbance of pancreatic digestion.

(*g*) Amorphous calcium bilirubin concretions in the biliary duct. Not recognizable clinically. Cause, so-called genuine stone diathesis.

If a gall-bladder affected with hydrops can be pushed towards the right, more easily than towards the middle line, it is almost always mistaken for a *movable kidney*, even by experienced observers. The difference can only be established by carefully determining the usual position of the swelling, and the ease with which it can also be moved towards the left. On the other hand, a hydronephrosis or a pyonephrosis may have grown so much towards the middle line and to the front that it can be felt and even seen in the gall-bladder area. Under these circumstances, the cystoscope is required before a diagnosis can be established.

(6) ACUTE CHOLANGITIS.

Some cases of incarceration of stone in the common duct, or of acute infective disease (typhoid, cholera, pneumococcic infection) are followed by an intensely septic state, and there supervenes a clinical picture of rigors, with high fever and jaundice. This indicates an acute cholangitis, which is caused by an ascending infection in gall-stone disease, and as a blood infection in the diseases just mentioned. Its course is that of an acute septicæmia and the patient rarely survives, even after early drainage of the gall-bladdder.

We assume that there is passive congestion of the bile when the fæces are colourless and the urine contains bilirubin, and that the jaundice is due to damage to the liver cells, when the fæces remain coloured and the urine contains urobilin exclusively or predominately.

CHAPTER XLIII.

TUMOURS OF THE LIVER.

It has happened that—on casual examination—a **movable liver** has been mistaken for an abdominal tumour. Careful examination will, of course, show that the liver is absent from its normal position, and that therefore the structure felt in the mid-abdominal region must be this missing organ. It is only necessary to place the patient in a posture wherein the thorax is lowered to observe that the liver resumes its normal situation. It may also happen that a liver, which is not especially movable, is pushed so far downwards by an extensive right pleural effusion that palpation suggests, for the moment, that an abdominal tumour is really present. Such an error is more conceivable in cases of a **constricted lobule** of the liver, especially if, owing to an aberration of the sense of beauty, the patient has undertaken self-treatment of the constriction, and has thus produced various derangements of digestion. The diagnosis is usually easy, and is indicated by the unnatural waist-like constricting furrow, which is visible on the skin, and by the fact that the lobule, which is felt, is connected with the liver. If the lobule is very movable, it is easily mistaken for a movable kidney, especially as it can sometimes be displaced into the renal region.

As a rule, it is quite easy to decide on careful examination whether a tumour-like structure is connected with the liver. If the tumour projects far downwards, the transverse colon may lie over it, and on percussion a zone of intestinal note will appear to separate it from the liver. The condition can, however, be cleared up, by examining the patient in varying states of intestinal fulness, or after artificial emptying of the colon. Tumours of the liver are sometimes simulated by large cancers of the stomach and occasionally by renal tumours which have grown forward.

Let us assume a case in which there is a localized tumour-like structure in a liver, which is not otherwise enlarged. The most probable diagnosis is a **secondary malignant growth,** and this view would be confirmed by a history of general malaise and wasting for some considerable period before the appearance of the tumour, or by the presence of several nodules. We may only assume that the case is one of **primary growth of the liver,** if an exhaustive examination has failed to reveal an original focus.

It is not possible to definitely distinguish between an innocent and malignant growth, before the onset of jaundice and cachexia. If it is possible to feel through the abdominal wall that the tumour is round in form and soft in consistence, it would suggest an innocent growth, cyst or cavernous angioma. But if the tumour is nodular, or if umbilication is detected, which is quite possible in thin subjects, then it is almost certainly malignant—**cancer.** But a **gumma** of the liver may closely resemble a tumour, and thus lead to errors ; therefore the serum test and specific treatment are indicated in all doubtful tumours of the liver.

If the patient is jaundiced the question of malignancy is settled, and therewith also the uselessness of surgical intervention. A **multilocular hydatid cyst,** referred to below, with its firm consistence and its somewhat nodular surface, may however be a source of error. If there are multiple growths in the liver we are justified in concluding that they are of a secondary metastatic nature, and therefore that they are malignant. The former conclusion is, however, not always accurate. There are primary **adenomata of the liver,** which are multiple and which permeate the whole organ with large nodules. These are usually accompanied by jaundice, and they behave clinically like malignant growths. **Cysts** which are quite innocent may also be present in some number.

This leads us to **cystic tumours** of the liver. If we find, in immediate connection with the liver, a structure which by its size, roundness of form, and soft elasticity of consistence, declares itself to be a cyst, we should not abandon further investigation and proceed to an exploratory puncture, in order to decide whether it is an **abscess**, a **hydatid** or some other **cyst.** The puncture will probably shed no new light on the diagnosis and may kill the patient. However fine the trocar, the fluid, if under pressure, and if the cyst is suppurating, may infect the peritoneum. Death has also followed rapidly, from the absorption of non-infected hydatid fluid. We must, therefore, endeavour to arrive at a diagnosis by other means. An example will illustrate the method.

A young man came from Bulgaria to our neighbourhood, which is free from hydatid disease. After four years' residence a tumour appeared in the hepatic region. The patient incidentally told the doctor that he had noticed a sudden onset of urticaria after a blow sustained over the tumour. The doctor argued as follows: Dogs play a much greater part in the life of Bulgaria than they do among us; therefore there is a greater probability of echinococcus infection. The blow over the tumour may have led to the absorption of some of its contents, and experience shows that a fluid whose absorption causes urticaria is hydatid fluid; therefore the tumour must be a hydatid cyst. The operation confirmed this conclusion.

Urticaria is indeed a very significant sign of absorption of hydatid fluid. But if no such indication is present, and the neighbourhood is free from echinococci, there is no other course to pursue but that of exploratory laparotomy. Sometimes it remains for the pathological anatomist to make a definite diagnosis.

The diagnosis of *multilocular hydatid* is difficult. If a patient is suffering from a very firm, irregular enlargement of the liver, with chronic jaundice, which even in the course of years causes no deterioration in the general health, the possibility of this variety of hydatid should occur to us if the condition cannot otherwise be explained. If the patient's habits have brought him into close contact with dogs this suspicion is confirmed.

But if there is no indication of hydatids, and, on the other hand, the patient has lived in the Tropics, or has suffered from gall-stones, we should think of **abscess of the liver.** This assumption is confirmed by intermittent or remittent high temperature, by rigors, pain in the right shoulder, pleuritic symptoms and increasing cachexia. The absence of these symptoms, however, need not make us discard the diagnosis of abscess. Liver abscesses may exist for a very considerable time **without fever,** so that the local swelling, the localized pain and the etiology must suffice to indicate the nature of the malady. But if these local signs are also absent, because of the deep situation of the abscess, the diagnosis is quite impossible and the condition may remain undetected for months.

A chronic hepatic abscess, in these latitudes, is very likely to be mistaken for cancer, because this latter is a much more frequent sequel of cholelithiasis than is a solitary abscess. In doubtful cases, an exploratory incision is advisable, because this may lead to the cure of an abscess and can do no harm in the case of cancer.

If a fluctuating swelling is found, in the absence of any history supporting either a hydatid or an abscess, we must assume that it is one of the rare forms of **non-parasitic liver cyst** (dermoid, ciliated cyst, cystic adenoma), the nature of which can only be ascertained by microscopic examination.

CHAPTER XLIV.

SURGERY OF THE PANCREAS.

We have already mentioned that the whole of the pancreas can sometimes be felt in thin people, with enteroptosis. This is a fact of importance, for the inexperienced are liable to mistake such a pancreas for a pathological structure. Diseases of the pancreas are not frequent, but those which can be diagnosed with reasonable probability are among the rarest incidents of surgical practice.

The varied symptomatology of these diseases may be classed under three headings, which we shall deal with briefly.

(1) ACUTE PANCREATITIS AND PANCREATIC HÆMORRHAGE.

If an individual—generally a male of advancing years—is attacked by symptoms which resemble either acute peritonitis or intestinal obstruction, often after suffering some indefinite prodromal intestinal discomfort, and if these symptoms are mainly localized in the upper abdominal region, we are justified in thinking of the possibility of pancreatic hæmorrhage or acute pancreatitis. This assumption is not negatived by slight jaundice, but it is supported by an early acceleration of the pulse and a lowering of the blood-pressure. If the initial, spontaneous and pressure pain is especially pronounced in the upper abdominal region, and if, after the cessation of the muscular rigidity, a diffuse resistance is appreciable in the epigastrium, the assumption becomes a probability. The rapid loss of strength and enfeeblement of the pulse, the early onset of low delirium and the possibility of evacuating gas by means of enemata, without relieving the patient or causing the vomiting to abate, are points against ileus. The limitation of rigidity to the upper abdomen and the early onset of meteorism are points against acute perforation of the stomach, although the seizure of pain may be just as sudden as in perforation. In one of my cases it occurred during defæcation. It may be some consolation for the practitioner who makes his diagnosis at the autopsy, to know that Germany's greatest surgeon recently succumbed to acute pancreatitis without anyone even suspecting the possibility of this condition. These diseases appear to be more frequent in some districts than in others. The fact that the cases sometimes come on as a result of cholelithiasis is not of much help to diagnosis, because any recurrence of disease in such patients would naturally be ascribed to an acute exacerbation of the old trouble.

Although the diagnosis can, as a rule, only be suspected or regarded as probable, nevertheless, the relationship between this disease and biliary disease does permit a more definite conclusion to be arrived at in certain cases. For example :—

A stout female patient, aged 50, was recovering from mild jaundice, associated with a gall-stone attack, when she was suddenly seized with severe pain in the upper abdomen, accompanied by collapse and followed by vomiting, retention of flatus and slight meteorism. In the course of the next twenty-four hours there was definitely increased dulness in both flanks. The pulse was rapid and soft. The pain was localized to the left of the middle line, thus contrasting with the pain of biliary colic. There were two possibilities; either perforation of the gall-bladder or acute pancreatitis. The position of the pain favoured pancreatitis, and this diagnosis was confirmed by the operation, which was performed forthwith.

In the rare instances wherein a patient survives the acute stage of pancreatitis without operation, a so-called pancreatic cyst may develop subsequently. We shall refer to this later on.

The presence of sugar in the urine is of diagnostic importance; but it is only found in a fraction of the cases (about one-fifth, in our experience), and then it is often very transient. A high anti-tryptic content of the blood is equally significant, but, unfortunately, the test takes too long to be of service in early diagnosis.

Cammidge's pancreatic reaction of the urine has hitherto not proved of much practical value in these diseases, because this test is not specific and requires an experienced chemist with a chemical laboratory. Neither is the examination of the stools for trypsin of clinical value. The presence of fat in the stools is only of significance when the bile is normal.

The practitioner who keeps in mind the possibility of pancreatitis, and takes the above mentioned diagnostic points into consideration, is in the best position to make a probable diagnosis, and give his patient the advantage of an early operation.

(2) CHRONIC PANCREATITIS: CANCER OF THE HEAD OF THE PANCREAS: PANCREATIC CALCULUS.

In a case of persistent jaundice, with stools which show a deficient absorption of fat and digestion of meat, and wherein there is also a very dilated gall-bladder, we are justified in thinking of some morbid change which is simultaneously obstructing the common bile-duct and the pancreatic duct.

If, in addition, a resistance can be felt in the epigastrium on the right, close to the spine, we may assume that the head of the pancreas is diseased. But we cannot tell from the clinical symptoms whether the disease is *cancer*, chronic *interstitial inflammation*, or a *pancreatic stone*. We cannot tell definitely whether it may not be a cancer of the common duct or of the duodenum, which is resembling disease of the head of the pancreas. It is sometimes very difficult or even impossible to make an accurate diagnosis, even at the operation.

Chronic interstitial inflammation may result from a gall-stone in the papilla of Vater as well as from a pancreatic stone. But it may also be of a specific nature—syphilis more frequently than tubercle. Chronic, sub-acute suppuration, or even gangrene, of the pancreas may occur owing to the presence of stones. If one thinks of this possibility in any given case, the diagnosis might be confirmed by a skiagram, as these stones mainly consist of calcium carbonate, otherwise it will be made, for the first time, at the operation.

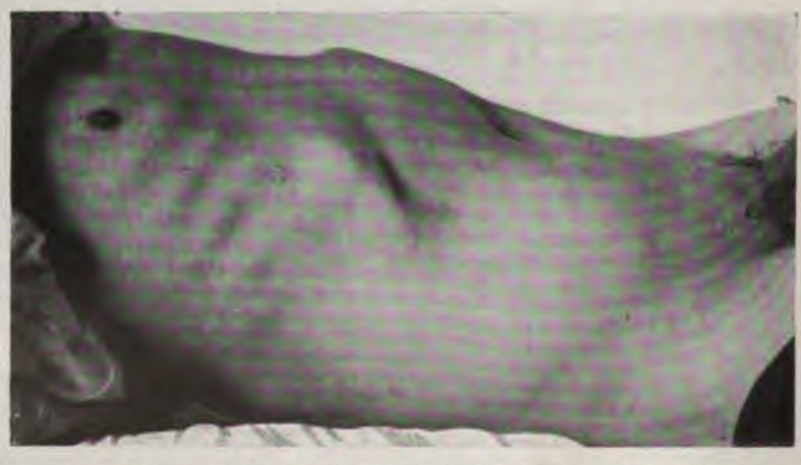


FIG. 190.—Inflammatory false cyst of pancreas, projecting between the ensiform process and umbilicus.

(3) PANCREATIC TUMOURS AND CYSTS.

A localized tumour in the mid-line of the epigastrium, between the stomach and transverse colon, but which can be shown to be independent of these organs by distending them with air, and which does not give rise to jaundice or any other striking symptom, suggests a pancreatic tumour which does not involve the head of that viscus. If the structure is not very large, but is firm or nodular, it is probably a malignant growth. But if it is larger and rounder, and more or less tensely elastic in consistence, it is a *cyst*. But if it has been preceded by symptoms of acute pancreatitis, we may assume its pancreatic origin with confidence.

A man aged 52, after suffering from a series of attacks of gall-stone colic, was seized with symptoms of acute pancreatitis, with 4 per cent. of sugar in the urine. The symptoms subsided, the sugar vanished, and a cystic structure developed in the epigastrium. In the course of 8 months it attained the size of a fœtal skull, and its situation was above the stomach. *Diagnosis*: Inflammatory false cyst of pancreas. This was confirmed by operation. Recovery.

We term this a *false cyst*, because it is not a genuine cyst lined with epithelium, but merely an effusion of blood, inflammatory exudation and pancreatic secretion within an encapsuled space of the sac of the lesser omentum, or in the retro-peritoneal tissue. Genuine cysts of the pancreas do occur, but they are much more rare.

CHAPTER XLV.

SURGERY OF THE SPLEEN.

THE spleen exemplifies especially well the present-day border line between medicine and surgery. The surgeon is left in undisputed possession of abscesses, genuine tumours and hydatids; but certain forms of splenic hypertrophy are still the subject of controversy.

(1) ABSCESS OF THE SPLEEN.

The previous remarks concerning abscess of the liver also apply to splenic abscess: its course may be quite free from symptoms, and remain unrecognized until it bursts into a neighbouring organ. If it is attended by symptoms, they consist of enlargement of the organ, pain on pressure, and also spontaneous pain, in the left hypochondrium. But another consideration is necessary before we can make a diagnosis, viz., the etiology, because splenic abscesses are apt to occur after typhoid fever, intermittent fever, or any pyæmic disease. But as the spleen so frequently enlarges in these conditions, we must not assume the presence of an abscess, unless the enlargement is greater than usual, and increases rather than diminishes after the subsidence of the original disease. Inflammatory symptoms in the vicinity, such as left-sided pleurisy and œdema of the anterior or lateral wall of the abdomen, are points in favour of the diagnosis. Puncture is as inadvisable here as in hepatic abscess, unless one is prepared for immediate operative treatment of the abscess.

(2) SPLENIC HYPERTROPHY.

The diagnosis of the various forms of diffuse enlargement of the spleen must be shared with the physician. He justly claims all enlargements due to *circulatory disturbance*, as congested spleen, the splenic enlargement of hepatic cirrhosis and the various forms due to portal obstruction. There is no surgical treatment for these, unless an attempt is made by means of Talma's operation. The splenic hypertrophy in all these cases is secondary to some other condition. Surgery is equally ineffectual when the hypertrophy is associated with a blood or lymph disease, such as leukæmia, pseudoleukæmia, or polycythæmia. Enormous leukæmic spleens have been removed, but the results have invariably been fatal. *Banti's disease* forms an exception, but we do not know whether this is a disease *sui generis*, and how it should be classified. Extirpation of the spleen has benefited some cases. The diagnosis can be made, if the malady has started with splenic enlargement and has been followed by slight jaundice, bile in the urine and cirrhosis of the liver.

Recent experience seems to show that removal of the spleen is also of therapeutic value in *pernicious anæmia*—whether the benefit is permanent is doubtful.

Infective and toxic enlargements of the spleen (*congenital syphilis* and *amyloid disease*) cannot be influenced by surgery. The *malarial spleen* forms an exception, for I have myself seen its removal followed by an unexpected recovery from a severe malarial cachexia. The diagnosis of a malarial spleen is not difficult, if we remember its tendency towards downward displacement. It can always be recognized by its sharp edge, even if it has sunk over to the right side. I have, however, seen a malarial spleen sent in for operation as an ovarian tumour, because its lower end was adherent in the true pelvis.

(3) TUMOURS OF THE SPLEEN.

If the splenic region is occupied by an irregular tumour which does not conform to the shape of the spleen at all, or only does so indefinitely, we are justified in thinking of a new growth, usually, as experience shows, a *sarcoma*. The confusion with a renal tumour may be avoided by remembering that the spleen can always be felt more definitely in front, while the kidneys are better felt from behind. This applies to the rare *serous cysts of the spleen* and to *hydatids*, which are also not very frequent.

A pronounced splenic tumour in a tubercular subject should suggest the possibility of a tubercular spleen, a condition in which surgery has effected some useful results.

CHAPTER XLVI.

ACUTE APPENDICITIS.

We have already seen in Chapter XXXVI how to recognize appendicitis, and to avoid the numerous diagnostic errors which may attend it. It now remains to determine what physical examination can teach us about the *condition of the appendix and its surroundings.* We shall confine ourselves to those facts which are recognizable with ease, and which are important in treatment. As long as one is quite clear about these, no blame will attach to him if he is in doubt about details of pathological processes in the appendix.

These chief points range themselves around the following four questions :—

(1) *Is the inflammation limited to the appendix and its immediate neighbourhood, or has it already extended beyond?*

We purposely say "and its immediate neighbourhood" because as long as the inflammation is exclusively confined to the appendix the clinical signs of an "attack" cannot have appeared. These only arise at the moment when the inflammation involves the neighbouring serous surface. This condition represents what is usually termed the early stage. The peritoneum is slightly reddened, and some fibrin is sometimes already found on the appendix. The abdominal cavity contains some early exudation in about half the cases. Our observations show that this exudation is already turbid and full of leucocytes on the first day, in about one-third of the cases ; but is nearly always sterile, or at any rate contains very few bacteria.

After the first day, the exudation is purulent in two-thirds of the cases, and is always richer in bacteria than on the first day. The odour usually becomes offensive on the third day, and the micro-organisms predominate over the leucocytes, while the exudation no longer retains the homogeneous saponaceous appearance which it had at first.

One is tempted to diagnose this early stage from the circumstance that the attack has come on within the last twenty-four hours. This would no doubt be true in most cases, but not in all, for we sometimes find signs of severe infection at the end of the first day, and even pus about the appendix. On the other hand, the disease may remain in the first stage for a considerable time, so that an "early" operation can be done after several days without any danger. The decisive point does not lie with the element of time, but with the result of physical examination. We must consider that the patient is still within the first stage of his attack *if the upper half of the abdomen and the left side are still soft and not tender, if there is no pain in the lumbar regions, at any rate the left, and if the pain on pressure and localized reflex contraction are limited to the suspected site of the appendix without any extensive dulness or definite resistance* (fig. 180 *a*). It is always wrong, and frequently dangerous to assume that the early stage lasts forty-eight hours. Whereas the mortality after operation on the first day is 0·7 per cent. it rises to 4 or 5 per cent. on the second day.

If we see the patient in what is really the early stage, it is our duty to propose an immediate operation, which then has every prospect of being a radical procedure.

If in spite of this advice the patient decides to accept the risk of a fully-developed attack of appendicitis, we can at least say, "*Dixi et salvavi animam meam.*" But we must be absolutely sure of our diagnosis in order to advise operation with a good conscience. The removal of an appendix in hysteria, muco-membranous colitis, typhoid fever, or even in pneumonia, does not redound to the credit of surgery.

(2) *Is an inflammation which has gone beyond the first stage localized or generalized?*

If the disease has extended beyond the first stage of strictly limited appendicular inflammation, and of early aseptic exudation, confined to the immediate vicinity, we have to decide how far the inflammatory process has travelled. *If the abdomen is everywhere sensitive to light percussion, if pressure on the lumbar regions elicits pain, and if palpation produces extensive reflex muscular contraction, it is quite certain that there is considerable involvement of the peritoneum, even if there is no dulness to be detected in the dependent parts. But if we can demonstrate an area of resistance which is sensitive to pressure, dull, or highly tympanitic on percussion, whereas the rest of the abdomen is comparatively soft, not at all or only slightly distended, and not sensitive to pressure, then the process before us is a localized one.* This localized process probably represents a fibrinous or fibrino-purulent inflammation of the neighbouring intestinal coils in those cases wherein the symptoms abate in a few days; and in those wherein the resistance persists for three or four days, or actually increases, it means a definite abscess.

(3) *Upon what does a general involvement of the peritoneum depend?*

In regard to this we must distinguish two conditions which are quite separable clinically, in typical cases. The case may be one of simple "early exudation," which will subside after the inflammatory area has become encapsuled.

This diagnosis should be made if *the attack is at its commencement, with pulse and temperature in correct relation to each other, if the patient does not look very septic, if the muscular rigidity is neither severe nor very extensive, and if the vomiting has ceased on the second day.*

The generalized inflammation may, on the other hand, depend upon an initial *severely septic infection* of the whole peritoneum. This is usually due to acute gangrene of the appendix, or to an extensive perforation which has flooded the peritoneal cavity with septic material. The exudation is very septic from the start, and, indeed, contains more bacteria than leucocytes. We base this diagnosis on the presence of *pallor, cyanosis, rapid thread-like pulse, normal or subnormal temperature in the extremities and in the axilla, but a high rectal temperature, dry tongue, and on semi-consciousness supervening on the second or third day, with a persistence of vomiting* (fig. 178, *i*). If these symptoms should appear during the later course of the disease, it means probably that an abscess which had been originally localized has burst into the abdominal cavity. In these cases it is still generally possible to discover the site of the original localized inflammation.

The cases of *sero-purulent peritonitis* are intermediate between those with mild, sterile, or slightly infective early exudation and those of severe septic peritonitis. Their diffuse symptoms of inflammation generally abate during the first week, but they leave infective germs in various portions of the abdominal wall, which are partially absorbed, but which also lead to the formation of localized abscesses—so-called *residual abscesses*—in the course of two or three weeks.

The differentiation of these various forms of general peritonitis is not merely of theoretical interest, but has important therapeutic bearings. If we or the patient have allowed the opportunity of an early operation to slip by, and the case is in the stage of early exudation of a mild character, we need not be anxious, but may await its subsidence with a clear conscience, and anticipate the localization of the process. But if a diagnosis of diffuse septic peritonitis has been made, the abdomen must at once be opened in several places—it must be washed out and thoroughly drained as circumstances demand. The younger the patient, the more likely is the issue to be successful. It is more difficult to recognize the intermediate forms, which often enough develop into localized abscesses even without our intervention. But, nevertheless, it is better to open the abdomen in the loins or in the middle line if necessary, to wash it out if adhesions are quite absent and to provide free drainage. This small procedure will often succeed in tiding the patient over a critical period—if combined with the application of warm compresses, subcutaneous and rectal infusions and the administration of stimulants.

Immediate operation must be undertaken if a fresh attack of acute general peritonitis starts from a localized area after the subsidence of the original diffuse symptoms.

(4) *What is the position of the localized abscess which we have decided to be present?*

The outlines of this position are at once determined by percussion and palpation. A glance at fig. 178 will show the chief positions which are involved. But there are other points to be elucidated in the interests of correct treatment, and these demand a careful examination. In the first place, we must decide whether the pus is intraperitoneal, retroperitoneal or even sub-fascial, *i.e.*, beneath the fascia of the iliacus muscle. If the abscess definitely projects into the abdominal cavity, it is intraperitoneal. If it fills the pelvic fossa and its shape is flat, we conclude it is intraperitoneal when there is pronounced reflex contraction of the anterior abdominal wall; we conclude that it is retroperitoneal if there is contraction of the flexors of the hip joint; and if this contraction is very pronounced, we conclude that the abscess is under the iliacus fascia. If the resistance is more in the lumbar region, we decide for an intraperitoneal position, if the illness started with definite signs of peritoneal irritation; but if not, we decide for retroperitoneal position, as also if a phlegmon appears in the lumbar region after a few days. Abscesses which track under Poupart's ligament are always sub-fascial. In doubtful cases, a temperature between 102° and 104° F., and rigors, point to an extraperitoneal position.

We must endeavour to determine the upper and lower limits of intraperitoneal abscesses, and also whether they are in contact with the anterior abdominal wall. If the lumbar region is painful on pressure, and is dull, and if the muscles are rigid, it signifies that the abscess reaches towards the kidneys, generally along the outer side of the ascending colon. If the patient complains of bladder trouble, the suppuration extends towards the true pelvis. If rectal symptoms are also present, and jelly-like mucus escapes from the rectum, the abscess is situated in Douglas' pouch. If the rigidity of the anterior abdominal wall is so great that the limits of the abscess cannot be clearly determined by palpation, we assume that it is directly in contact therewith. If, on the other hand, its limits are very easily defined, there is probably a free peritoneal space between the abscess and the anterior abdominal wall. If meteorism is present, an abscess deeply situated between the loops of the small intestine (meso-cœliac) may completely elude palpation. Unless we have watched such a case from the beginning, we are liable to diagnose either a generalized peritonitis or an ileus. The latter mistake is all the more pardonable, because such an abscess occasionally causes genuine obstruction by compressing or kinking the bowel.

The correct determination of the position of the abscess is important in order to decide *whether* and *how* to operate.

If we see a patient on the third or fourth day with the inflamed area in process of encapsulation and probably already subsiding, we should do nothing lest we disturb the natural defences of the body, or rather maintain an "armed peace," because the process may burst forth anew, and again become dangerous.

If the general and local symptoms show no sign of subsiding at the end of the first week, or if, indeed, they increase, an operation must be performed without any further delay, not so much for the purpose of removing the appendix, but in order to evacuate the pus and thus avoid the immediate danger. We should act in the same way if a primary or secondary collection of pus forms anywhere during the second or even the third week. No special scheme of operation should be followed, nor should any particular incision, recommended by one surgeon or another, be adopted, but the abscess should be opened at its most superficial situation and with as little anatomical damage as possible, i.e., in the ileo-cæcal region, lumbar region, linea alba, or finally in the vagina or rectum. Large incisions are not only unnecessary, but they are responsible for the subsequent development of hernia in the scars. It is therefore important to find, by means of a careful local diagnosis, the point where the smallest incision will suffice. If the pus is behind the peritoneum, we must approach it from the side. If it is deeply situated, in the meso-cœliac area, separated from the abdominal wall by the free peritoneal cavity, we should not hurry quite so much with its incision and, if necessary, must carefully protect the free abdominal cavity during the operation.

We have not yet said anything about *exploratory puncture* in the diagnosis of appendicitis. Careful observers, and those who have had opportunities of witnessing the topographical condition during operations for appendicitis, will not feel the want of this method. As Roux says, "It is not always free from risk, is often useless and is always unnecessary." It is only useful in subphrenic or pelvic abscesses which are more or less inaccessible to direct examination, and then it is indispensable.

We have hitherto had in view the pure, typical attack. But if, either through our own fault or that of the patient, he has arrived at a stage when we no longer have full control over the disease, we have to reckon with several complications which will make demands upon our diagnostic skill. We will assume that the initial peritonitis, as such, has been overcome, that the abdomen has become temporarily softer, the respiration quieter, and the temperature normal, or approximately so.

One group of complications is indicated by the fact that the improvement stops short at a certain point, and that the temperature rises again and takes the form of an abscess chart. In such a case it is very probable that germs diffused throughout the abdominal cavity are developing into *residual abscesses*, the favourite positions of which are indicated in fig. 178. Experience teaches us that we should look for them in *Douglas' pouch*, and in the *left flank* during the first and second week. An abscess in Douglas' pouch can at once be recognized by rectal examination. If we have neglected to search for it in time, our attention will be drawn to its presence by a discharge of mucus from the rectum, by tenesmus, and sometimes even by failure of the action of the bowels. Occasionally, we may be taken by surprise, by the spontaneous rupture of the abscess into the rectum or vagina.

An abscess in the *left flank* is easily recognized by local pain on pressure by muscular rigidity and by dulness. We should also think of the *right flank* if the case has not been operated on, or if the drainage in the right lumbar region is inadequate.

After the third week a *subphrenic abscess* is the most likely complication. It rarely comes on earlier, and is usually situated on the right side. If nothing abnormal is found in any of these positions, and the abdomen still refuses to become soft, it is probable that small residual abscesses exist between the coils of the intestines. It is best, as a rule, to allow these abscesses to become spontaneously absorbed, because the search for them is liable to do more harm than good.

In other cases the complications are indicated by colic, followed by vomiting, either very rapidly, or within a few days. The temperature remains normal, or is only slightly raised, and the patients appear to be quite well between the attacks of colic. These cases certainly depend upon a partial or complete *intestinal obstruction*, due to adhesions and kinking of a coil of intestine. These complications usually occur in the fourth to the sixth week, and sometimes earlier. Experience shows that this obstruction has a greater tendency to disappear spontaneously, the earlier it sets in. This should be borne in mind as a therapeutic indication.

In contrast to this early obstruction, there is another form which may come on, after months or years, and which is due to narrow bands of connective tissue. Immediate operation is required in this variety.

If we unexpectedly find the patient very feverish, with flushed cheeks and rapid shallow breathing, and with a dicrotic pulse, we should at once suspect a respiratory complication, either a metastatic pneumonia or a pleurisy. In the latter case, we may be quite certain that it arises from a concealed focus of inflammation in the lung, or from an undetected subphrenic abscess—the latter especially if the pleurisy is on the right side.

Among other "unforeseen" complications which occasionally occur in appendicitis may be included, phlebitis, parotitis, and other inflammatory processes which can easily be localized.

Much anxiety is often caused, both to the medical attendant and the patient, by the long persistence of sinuses after the opening of abscesses. This is almost always due, either to the tubercular nature of the disease, or to a fæcal concretion which has escaped from the appendix into the abdominal cavity, but which has not yet been discharged in the pus.

The foregoing remarks apply to the cases wherein it is possible to diagnose the *onset of appendicitis* with certainty. In the *intermediate* stage, however, the diagnosis is much more difficult, often, indeed, impossible. If a severe case of appendicitis happens in a family, the other members devote considerable attention to their own ileo-cæcal regions, and anxiously consult their medical adviser as to the propriety of removing the appendix; they complain of stabbing, burning, or dragging pains in that vicinity, and feel that it is very sensitive to pressure. With the very best of intentions, the doctor can detect nothing; his diagnosis must be that of a hysterical or imaginary appendicitis, and he sends the patient home. He will generally be right, but not always, and therefore should not entirely lose sight of the patient.

I refused to operate, under these circumstances, on a young man upon whose sister I had just operated, but advised him to report himself if he really got a genuine attack of pain. He did so a few weeks later on. I operated at once, and found a highly inflamed appendix, with a commencing fibrinous peri-appendicitis.

What criteria can guide us in these circumstances? *History* is of the first importance. If the patient has previously had an "inflammation of the bowels," which can be designated as an attack, or, better still, if a definite antecedent attack has been reliably observed by a doctor—not a mere abdominal pain lasting a few hours—the patient is correct, and we must accede to his wish.

But if the ileo-cæcal, gastric, or more diffuse pains are indefinite, and do not keep the patient in bed, we must reserve our verdict. It may be a form of appendicitis which has not yet developed a "first attack," called by Ewald "larval appendicitis." But, as Gussenbauer has remarked, it is only larval in the sense that the doctor has not been able to diagnose it. But in this connection the remarks made in Chapter XXXVI should be recalled.

The *physical condition* will often fill in the gap. In the first place we must endeavour to exclude any other abdominal disease, especially in women. Then we should palpate the normal situation of the appendix. Daily experience of operations during the free interval teaches us that this is not at the point where the outer border of the rectus meets the line joining the anterior superior spine with the umbilicus, which, according to McBurney, marks the orifice of the appendix. It is the ileo-cæcal valve, which is usually situated at this spot. The position of the appendix is more simply and more certainly ascertained by marking the spot where the line joining the two spines cut the outer border of the right rectus (Sonnenburg). If nothing abnormal is found here, or in any of the other situations usually accredited to the appendix, we still remain in doubt; and if the history is indecisive, we must await events. If the history is definite, however, the negative result of our physical examination is of no significance. If there is a complaint of moderate pain upon pressure, but no change can be detected by palpation, we must rely upon the history, because a certain degree of sensitiveness to pressure may exist in normal circumstances. But if this pain on pressure is very pronounced, and much greater than the pain caused by pressure on the left side, we may assume that some chronic inflammation is probable, bearing in mind the possibility of chronic colitis (see next chapter). If, finally, we discover in a patient with a slightly flexed hip a sensitive structure, which can be distinctly rolled under the fingers, we must regard it either as an appendix or an adherent mass of omentum bowel and appendix, and should recommend operation to the patient.

We have already seen that it is quite exceptional to be able to palpate the normal appendix, and even if it has once been inflamed it can only rarely be felt during the free interval.

CHAPTER XLVII.

COLITIS, SO-CALLED CHRONIC APPENDICITIS AND FUNCTIONAL DISTURBANCES OF THE LARGE INTESTINE.

SEVERAL different conditions are grouped together under the terms colitis, chronic appendicitis, pseudo-appendicitis, &c. They are not all really examples of inflammatory processes, but they are classified together because they apparently merge into one another by means of borderland and transitional forms. The study of these cases convinces us that a true appreciation of them can only be obtained by differentiating those due to organic causes from those originating in functional disturbance. We therefore distinguish:—

A.—COLITIS WITH DEFINITE ANATOMICAL CHANGES.

This includes all the cases of inflammation which are recognizable by definite histological changes, and which usually terminate in ulceration of the mucous membrane. As these cases generally come to the surgeon in the stage of ulceration, they may be grouped together under the term of colitis ulcerosa.

How are these to be recognized ? Mainly by the great frequency of the diarrhœa, by the admixture with mucus and occasionally with blood, or at least with traces thereof, and also by tenesmus, if the disease affects the lower section of the large intestine. The physical signs consist of the rigid contraction of some intestinal segments, or their inflammatory infiltration. The symptoms are much less definite if the first part of the large intestine is alone involved, because the longer course of the intestinal contents may enable them to regain their normal state. There is not always diarrhœa, in these cases, and the symptoms are usually limited to vague pains, or to definite attacks in the ileo-cæcal region, which are usually diagnosed as appendicitis.

We must first satisfy ourselves that this colitis is not a manifestation of a more grave disease, such as intestinal cancer, or of polypus of the large intestine, which latter is very rare. It is a serious matter for the patient if he is treated for weeks and months without examination of the large intestine, merely for mucous and bloody evacuations, while he is suffering all the time from a rectal cancer, which could easily be felt. It must also be very annoying to all taking part in the case if it is allowed to go on until the surgeon finds the growth firmly fixed in the true pelvis. Every diagnosis of colitis invariably demands a rectal examination. If nothing abnormal is found, the sigmoidoscope must be used, and the whole of the large bowel must be palpated for a tumour, unless it is quite clear from the history and the onset of the disease that it has some other origin.

Having excluded, as far as possible, a new growth, and naturally also a pelvic abscess irritating the bowel, we must next determine the variety of the *colitis*. We can only tell for certain by means of the sigmoidoscope whether the case is chiefly of an ulcerative character. A skiagram may very probably give some information on this point. As already indicated by the diarrhœa, an ulcerated intestine endeavours to evacuate its contents as quickly as possible, or, to put it scientifically, exhibits increased motility. Stierlin has demonstrated in our clinic that this peculiarity is shown in the skiagrams by the rapidity with which the bismuth meal is hurried along the affected portions of the bowel. Indeed, they are always found empty or, at any rate, exhibit only a few bismuth stains, when a series of plates is taken. The border line between the bowel which is pathologically empty and that which is normally filled, is so sharply defined that the comparative examination of several plates usually gives an accurate idea of the extent of the disease.

It should be added that the bowel behaves in the same way, if it is infiltrated with new growth or tubercle, and not ulcerated. The injection of a bismuth emulsion will show whether the emptiness of a segment of bowel is due to reflex hyper-motility (ulceration) or to rigid infiltration—with or without ulceration. In the former case, the large bowel will be seen to possess its normal folds; in the latter case the folds are deficient. But if the ulcerative colitis is very severe, the bowel is also rigid, and is not thrown into folds by the bismuth injection. Fig. 191 is a typical illustration of this. If the infiltration extends to Bauhin's valve, it becomes functionless.

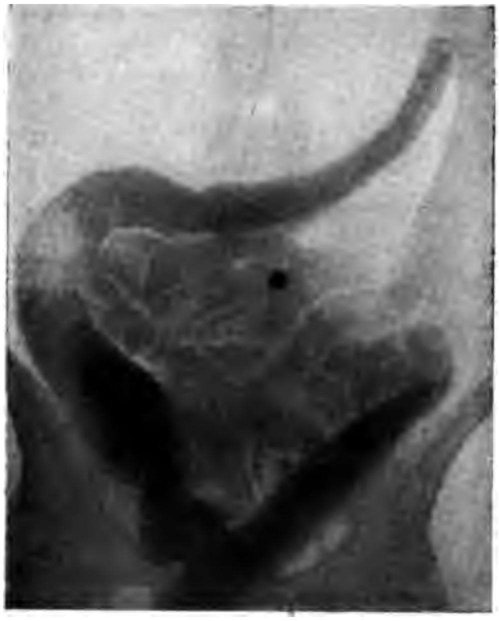


FIG. 191.—Severe case of ulcerative colitis, after a bismuth injection. Folds of large intestine imperfect. Appearance somewhat speckled. Bauhin's valve ineffective. (The injection fills a considerable portion of the small intestine).

Having recognized the ulcerative character and the extent of the disease, we have now to determine its nature. Is it due to *syphilis* or *tubercle?* Is it the late stage of an *amœbic enteritis*, a *dysentery caused by the Shiga-Kruse bacillus or the Flexner bacillus?* Is it a case of *chronic ulcerative colitis of unknown origin*, affecting a localized portion of the large intestine or its whole extent, of a type to which attention has recently been drawn?

Let us take **syphilis** first. This is usually limited to the lowest portion of the large intestine, and produces the well-known clinical picture of rectal syphilis, which we shall refer to later on. It rarely extends higher up into the bowel. Our diagnosis will be supported by the history, Wassermann's test and by the presence of other signs of old syphilis.

Tubercle is much more important. The diagnosis will be suggested by a tubercular heredity and previous history, the chronic start of the malady, and by other existing foci of tubercle. A positive diagnosis can only be made by a histological, or, if necessary, a bacteriological examination of a piece of the tissue taken from an ulcer with the aid of the rectoscope. The discovery of tubercle bacilli in the stools is of value, but it does not decide the diagnosis, because the patient may be suffering at the same time from pulmonary tuberculosis.

An **amœbic** or **bacillary dysentery,** in their late stages, will be suggested if the patient comes from a district in which these diseases are endemic. Physical examination does not elucidate matters in these late stages. The serum test is not very reliable, and only applies to the Shiga-Kruse bacillus.

If there is nothing pointing to any of the above-mentioned forms, we may assume that the case is one of **chronic ulcerative colitis,** of unknown origin, which in its severe forms may affect the entire large intestine. (Fig. 191).

It is however more frequently limited to the cæcum on the one hand and the sigmoid on the other—sometimes referred to as **ulcerative typhlitis** and **sigmoiditis.** This localization is explained by the fact that the fæces are retained longest in these positions and therefore they become *loci minoris resistentiæ.* The sigmoid is also particularly liable to localized disease in women, owing to its unfortunate proximity to the internal genital organs with their infirmities.

Graser's diverticula (multiple indentations of the mucous membrane beneath the serous membrane through vascular laminæ in the muscular layer) constitute one of the most frequent causes of severe acute and chronic sigmoiditis. These small diverticula can be seen in a skiagram in favourable cases (vide next chapter), and if they are low down can be demonstrated with the rectoscope.

If the inflammation involves the serous surface, in an acute or chronic manner, the conditions of peri-typhilitis (independent of the appendix) or peri-sigmoiditis are produced. Sometimes a peri-colitis of this nature proceeds even to suppuration.

From the the foregoing remarks, it is obviously superfluous to represent peri-colitis as a separate disease. It is merely the sequela of a colitis, which is often overlooked clinically, and is not a separate morbid entity.

B.—FUNCTIONAL DERANGEMENTS OF THE LARGE INTESTINE, WITHOUT TYPICAL ANATOMICAL CHANGES.

The more names a disease possesses, the less definite is our knowledge of it. **Muco-membranous colitis,** with which we shall mainly deal in this section, has received at least a dozen different names during the last thirty years. This disease embraces all the functional derangements which may affect the large intestine: *motor derangements*, in the form of *constipation* alternating with *diarrhœa ;*

sensory derangements, in the form of regularly recurring *colic* or severe seizures thereof ; *secretory derangements* in the form of persistent or occasional copious *discharge of mucus*, either in a glairy condition, or as coagulated masses of a tape-like or tubular shape. Clinical experience shows that this symptom-complex may come on after some psychical disturbance, may last for months or even for years, and then disappear as a result of mental excitement. But this symptom-complex may also represent the reaction of the large intestine to various pathological conditions, both within and without itself, ranging from cancerous, tubercular or other ulceration to displacements of the large intestine and inflammatory processes in its vicinity. It may also be due to toxic causes, such as alcohol, tobacco and mercury, and also to bacterial poisons.

The latter circumstance explains the erroneous conception of muco-membranous colitis, as the result of an intestinal infection.

The entire large intestine does not invariably exhibit this abnormal reaction, nor are all the three previously mentioned forms of functional disturbance fully pronounced. As a rule, only the motor, and, to some extent, the sensory disturbances are in evidence, while the secretory derangements are either absent or not striking. The condition is then essentially one of **painful constipation.** This leads to the question of the *localization of the functional derangement*, which may be solved by X-ray examination.

If a meal, consisting of 200 grm. of carbohydrate porridge and 80 grm. of barium sulphate, is given to a normal individual, the whole of it will be found in the cæcum, ascending colon and to some extent in the beginning of the transverse colon, after six to eight hours. The ascending colon is empty, or nearly so, after twelve to eighteen hours, and the contents are visible in the lower section of the large intestine. The whole intestinal canal is empty after twenty-four to thirty hours. If digestion in the large intestine is slow, the delay may be distributed over the whole of the large intestine, or it may occur exclusively in that section wherein the fæces remain longest in order to become inspissated, namely in the first portion as far as the level of the gall-bladder (Stierlin's ascending type of constipation. The delay may also occur in the last section of the large intestine (sigmoidal and proctogenic constipation).

In our experience the *ascending type* of constipation gives rise to most subjective symptoms. This is due to the fact that the intestinal contents in this position are still semi-liquid and are more prone to cause fermentation with the development of gases than the more or less dry fæces retained in the sigmoid, or the ampulla.

All this prepares us for the conception of a clinical picture, which has been described in France for the last fifteen years under the terms of *typhlocolite* or *typhlite ptosique*. It has only generally been recognized in Germany quite recently as *cæcum mobile* (Wilms), *typhliklasie*, *typhlatonie*, &c.

In the year 1897, shortly after the appendicitis rage set in, there was a great tendency to ascribe all ills in the ileo-cæcal region to the appendix, and the old idea of stercoral typhilitis was entirely discarded. At this time, however, Dieulafoy, who was himself an advocate for early operation in real appendicitis, pointed out that there were attacks of pain in the ileo-cæcal region which had nothing to do with the appendix, but which signified the localization of the muco-membranous colitis in the cæcum. Although names and theories have undergone much change since then, or rather have been considerably multiplied, no real advance has been made upon the position defined by Dieulafoy.

The established facts, as far as they possess diagnostic importance, may be summarized as follows:—

There are many persons, mainly females, who complain of their cæcal region, but who never get real attacks of appendicitis, lasting days or weeks, with the development of resistance or peritoneal exudation. The so-called "attack" usually resolves itself into a severe seizure of pain about the cæcum, which only lasts a very few hours and has generally disappeared by the time the doctor arrives. But if the patient can be examined during the attack, it will be found that tenderness exists in the ileo-cæcal region on pressure, but that there is no muscular rigidity. Indeed, a structure like an elastic cushion may be felt, and sometimes this vanishes under the hands of the examiner; but if it can be carefully palpated it will be recognized as the distended cæcum. Exceptionally, the temperature may be somewhat raised, and patients with active reflexes are liable to vomit. Sometimes the attack terminates in looseness of the bowels, at other times in diarrhœa. Then the tenderness on pressure also disappears; no localized pain remains over the appendix for a few days, as is the case even after a mild appendicitis. On the other hand, we do find, as Dieulafoy has pointed out, painful, contracted portions of the large intestine, indicating a state of abnormal irritability or chronic colitis, and, occasionally, the onset of mucous discharge confirms the diagnosis.

The cæcum can often be felt to be movable and capable of being displaced hither and thither. A skiagraphic examination of intestinal function will show that the contents are unduly delayed in the cæcum —*i.e.*, constipation of the ascending type.

Many of these cases have been submitted to operation, some under the mistaken diagnosis of appendicitis; others were diagnosed correctly and were operated on, because the disease was interfering with the nutrition of the patients and their ability to do their work.

The anatomical conditions found, consist of a large cæcum extending low down, a normal appendix, remains of inflammatory adhesions on the cæcum itself, and the so-called peri-colonic veil (which is merely the stretched mesenteric attachment caused by the tilting of the cæcum) on the ascending colon, reaching as far as the hepatic flexure. These changes do not, however, explain all the symptoms, even if we include the kinking of the small intestine in front of Bauhin's valve, as recently described by Lane. At least every tenth person has an abnormally movable cæcum, and very frequently adhesions and kinks produce no symptoms at all. Delay of the fæces in the cæcum also occurs without symptoms. We must, therefore, revert to our original view that functional causes play the chief role in all these troubles of the large intestine. A normally innervated large intestine overcomes all possible difficulties, and even resists the effects of abnormal conditions of nutrition and life generally. But if the innervation of the bowel departs from the physiological standard, it reacts towards abnormalities in the mode of life and slight mechanical difficulties, by simple constipation or by more or less definite symptoms of muco-membranous colitis.

The main surgical interest of this disease concerns its diagnosis. The practitioner must recognize it as a disease in itself, which, especially in women, is capable of mimicking all possible painful affections of the abdominal cavity, and which makes neurasthenics fearfully apprehensive of cancer, but he must also appreciate the important fact that this disease may be a concomitant manifestation of genuine cancer. It also has surgical interest from the point of view of treatment, because attempts have been, and are still being, made to cure the very severe forms of the disease by procedures, varying from a simple cæcostomy to extensive resections of the bowel, as no prospect of recovery is offered by medical treatment, including baths, diet, electricity or psycho-therapy. But it cannot be said that surgical treatment is always more successful than medical. It is sometimes quite impossible to restore the intestinal reflex actions to a normal condition, in a neuropathic individual, in whom all reflexes are out of gear.

We are now in a position to state what we mean by the rather inappropriate expression, **chronic appendicitis.** Logically, the term should only be applied to that form of appendicitis in which the appendix does not recover from its inflamed condition. This is especially the case with tubercular *appendicitis*, but this condition requires no new name. It also occurs in very many ordinary attacks of appendicitis, wherein the complete subsidence of the inflammatory symptoms is prevented by fæcal concretions, stenoses, adhesions, and kinks. In some cases the owner of the appendix is unconscious of this chronic irritation ; in other cases he suffers frequent but slight pains, reflex disturbances of the intestinal function, and even from muco-membranous colitis. Other patients only experience an indefinite discomfort in the right side of the abdomen. As most appendices which have suffered from several attacks remain in a state of chronic irritability, there is no object in separating them clinically from those cases in which this irritation becomes more pronounced than usual. This constitutes one class.

The other class has nothing at all to do with the appendix. It embraces those cases which were formerly justifiably termed stercoral typhlitis, and consists of localized ulcerative colitis, localized functional disturbances of the large intestine in their various forms, such as "typhlo-colite," cæcal distension, &c., either of mechanical or functional origin. It is obvious that these cases ought not to be called chronic appendicitis.

We would not have gone into this matter so much in detail were it not that it possesses great practical importance.

When may the practitioner be satisfied with the assumption that the case is one of mere functional disturbance of the cæcum, and therefore abstain from operation during the first twenty-four hours ? He is justified in this course if the attacks are frequent and of short duration (only a few hours), if there is mucous discharge and a pronounced neuropathic history, and if the actual attack subsides within a few hours. This affords adequate time for observation, so as not to miss the opportunity of early operation if the case is one of genuine appendicitis. In this connection it must be emphasized that pulse and temperature are in no way decisive, because we have seen cases of appendicitis with pus formation wherein the pulse has not exceeded 100 per minute and the temperature has not been above 99·5. On the other hand a functional derangement may be associated with a quicker pulse and a higher temperature. A definite leucocytosis indicates appendicitis, but this is often absent at the beginning of the disease. If the patient has not recovered at the end of the first twenty-four hours it is our duty to propose operation. It is to be hoped that the recently acquired knowledge regarding the cæcum and the ascending colon will not increase the mortality from appendicitis through neglect of operation.

CHAPTER XLVIII.

INTESTINAL OBSTRUCTION.

ONE of the most grateful tasks which can fall to the lot of the surgeon is to relieve a malady which is popularly recognized to be attended with great suffering. Early diagnosis and an accurate appreciation of the moment to interfere, are, however, essential for this purpose. Cases of intestinal obstruction exemplify better than any other condition how dangerous it is to wait for the fully developed clinical picture before arriving at a decision. To pursue this course is to sacrifice the life of the patient to refinement in diagnosis, excellent though the motive be. There is no object in being able to proclaim at the autopsy that we had correctly diagnosed the situation and nature of the obstruction. Our main object must be to recognize when surgical relief should be afforded, although we may not always know the precise position and character of the obstruction. This is no encouragement, however, to laxity in diagnosis. On the contrary, *careful observation, thorough examination*, and a *consideration of all signs* are indispensable, but this must be done *rapidly*, and we must *decide rapidly*, if our reflections are to be of any use to the patient.

In practice it is necessary to distinguish two great groups of intestinal obstructions: (1) A *complete* form which comes on *suddenly;* and (2) a form which comes on *gradually* and which is *incomplete* while it is developing into *chronic obstruction*. We will deal first with the latter, in which the process can be followed more leisurely in all its details.

I.—STENOSIS OF GRADUAL DEVELOPMENT.

(CHRONIC INTESTINAL OBSTRUCTION.)

A.—SYMPTOMS.

Colic, i.e., the painful contraction of a portion of the intestine is the first symptom of narrowing in its lumen. But colic is of such frequent occurrence that it does not signify very much by itself. But if pains of the same type regularly recur in the same area of the intestine they definitely point to a local trouble in the shape of a local obstruction of the intestine. This, however, is not sufficient for a diagnosis. Such pains may be present in **colitis** of any origin.

In order to confirm such a condition it is necessary that there should be abnormal **dilatation** and visible or palpable **contractions** of the bowel above the site of obstruction. The dilatation is recognized by the repeated occurrence of a highly tympanitic or metallic note, occasionally by the metallic sound of the peristalsis at the same site, and by spontaneous crepitant noises. Abnormal contraction is recognized by the periodical *hardening of the bowel*, which is very different to the contraction of the intestine in colitis. In the latter case, the bowel, which contracts when it is empty or contains a little fæces, feels like a firm band ("corde colique" of the French), whereas the hardened intestine—either the large or small—above an obstruction gives one the impression of an elastic tumour. That we are dealing with rigid intestine is quite clear from the tympanitic note on percussion, and from the periodical onset and disappearance of resistance. If we are fortunate enough to detect a **buzzing sound at** this spot as the resistance disappears, there can be no further doubt about the existence of a constriction. This applies not only when one coil of intestine hardens and relaxes with a heaving movement, but even when a whole segment of bowel shares in this process—when, as sometimes happens, as many as four parallel tumid coils are present (fig. 192). It is often necessary to wait and watch for some considerable time at the patient's bedside in order to witness these phenomena, if they cannot be elicited by palpation of the bowel. As the illness progresses and the compensatory hypertrophy of the intestinal muscle is no longer able to overcome the obstruction, consequent disturbance occurs, which declares itself by persistent abdominal distension, leading to complete intestinal paralysis. But this distension is by no means a *sine qua non* for the diagnosis of stenosis of the bowel, as beginners often imagine. There may be ileocæcal stenosis too narrow to admit a cherry-stone, and, nevertheless, the abdomen may be quite flat, or even depressed.

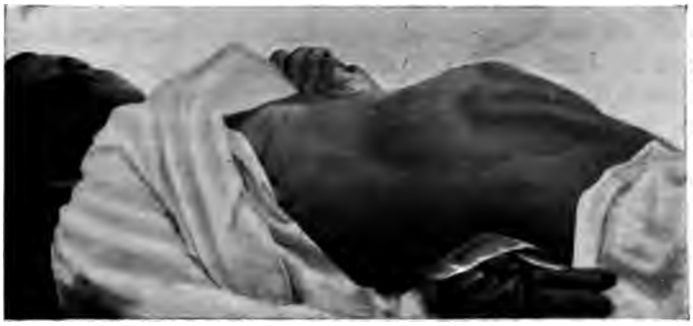


FIG. 192.—Rigid contraction of bowel, through obstruction of small intestine by a fibro-sarcoma.

The above-described symptoms are most pronounced in *stenosis of the small intestine.* The hardening in the *large intestine* is often very indefinite, which accounts for so many late diagnoses. This hardening can very frequently still be detected in stenosis of the ascending colon or hepatic flexure, but then it also involves the lowest coils of the small intestine after Bauhin's valve has been thrown out of action.

One sign, which we meet with in acute complete obstruction, is almost completely absent in incomplete cases, i.e., **vomiting.** It occurs first when the obstruction becomes temporarily or persistently complete, and the higher up the obstruction is, the earlier it takes place.

The condition of the **stools** affords important information, but not quite so much as is generally supposed. One often hears the verdict—there is no obstruction because the patient has normal stools ; or, on the other hand, that there is obstruction because the fæces are in little masses, looking like sheep's fæces. The one conclusion is as erroneous as the other. The stools form themselves slowly in the large intestine from the transverse colon onwards. If the obstruction is in its upper part, where the fæcal contents are normally liquid, there is nothing to prevent the normal formation of fæces below the obstruction. The patient, therefore, continues to have regular well-formed stools—often up to the moment when complete obstruction comes on. Even if the stenosis is in the neighbourhood of the splenic flexure, the fæces may still be formed normally. But when changes in the stools ensue as a result of more deeply situated stenosis of the large intestine they take the form neither of sheep's fæces nor of "tape," but of alternate complete retention —still euphemistically called constipation—and the evacuation of pulpy, soft fæces. This means that the intestinal contents above the stricture are not solid, "inspissated," but are pulpy or putty-like ; the narrower the stricture the more liquid they are. If in a case of obstinate constipation we meet with fæces of the shape of balls, even very small balls, there is no occasion for anxiety, for we may safely assume that this is due to mere sluggishness of the bowel, which leads to abnormal inspissation of the intestinal contents, in contrast to what occurs in stenosis.

In a case of mine, the medical attendant justly suspected cancer on account of persistent diarrhœa, the patient being an old man. The tumour was not palpable from the abdomen, but I discovered it at once on bi-manual recto-abdominal examination. It was situated below the sigmoid.

The so-called "tape-like" fæces occur when they are of clayey consistence, and have squeezed themselves through a constriction near the anus, within reach of the finger—but never higher up.

Tenesmus is always present in these cases (see "Difficulties in Defæcation"). In every case of "*constipation*" we must ascertain its duration. If it has existed for years there can be nothing seriously wrong; but if it has only been present for a few months in a person who had not been constipated previously it is a serious symptom and requires careful examination.

A man, aged 50, consulted his medical attendant because of recent constipation. He found nothing in the abdomen and prescribed fruit. The patient continued to eat fruit with the greatest diligence for four months, and, eventually, came with cancer of the rectum, situated so high up that it was hardly operable.

The conclusions to be derived from stools *mixed with blood* are only relative. If blood is present in a case already suspected of cancer, the suspicion is thereby strengthened. But we must not forget, that any ulcerative colitis may be accompanied by the passage of blood. Blood may also be present in muco-membranous colitis. In these cases the hæmorrhage is seldom profuse, but the masses of mucus contain within them specks of blood which lead to a correct diagnosis. The rectal mucous membrane sometimes bleeds when there are neither ulcers nor internal piles, the only change being one of hyperæmia.

An *intermixture with pus* indicates a deep ulcerative process in the lower part of the large intestine, such as may occur with a large excavating cancer or dysenteric ulcers. If the cancer is situated higher up, the pus is so intimately mixed with the stools that it is no longer separately recognizable. Any considerable evacuation of pus per rectum signifies that an abscess has broken into the bowel.

The significance of *intermixture with mucus* is not very great, because this occurs whenever the large intestine is irritated, in idiopathic colitis, as well as in colitis which follows tubercle or cancer.

We must be very careful in drawing any diagnostic conclusions from the **effect on the general condition**, because if the compensation is satisfactory, nutrition does not at first suffer. If the attacks of colic are frequent, the patient instinctively diminishes the amount of his food, and therefore emaciates, even if there is no persistent constipation. The question of emaciation is not decided by the amount of fat which the patient retains, but by the amount which *he has lost*. It is enough to pinch up a fold of skin, especially over the abdomen or thigh, to show us what was there before. If the relative obstruction has persisted for some time, there will always be a certain amount of cachexia.

B.—THE POSITION OF THE STENOSIS.

The site of the obstruction is sometimes indicated forthwith by rectal examination, by palpation, or by the position of the rigidly contracting loop of bowel. Sometimes it can only be determined by taking into consideration the points which will be advanced in the discussion of acute obstruction. But one must always remember that the signs of chronic obstruction are not, at first, very pronounced, because the obstruction itself is incomplete. We shall subsequently refer to the importance of the skiagram in diagnosing the locality of the obstruction.

One important peculiarity, which may give rise to error, should also be mentioned. Wherever the obstruction may be in the large intestine, e.g., in the sigmoid, the maximum dilatation of the bowel will not be immediately above it, but it will always be in the cæcum, as long as Bauhin's valve is in working order. The explanation, which can be inferred both experimentally and mathematically, is due to the double factor that in the ascending colon and cæcum the diameter of the bowel is greater, but the thickness of its wall is less than in the lower parts of the large intestine. This also explains why the most numerous and the deepest ulcers, which follow the dilatation, are to be found in the cæcum and ascending colon, even if the obstruction is at the lower part of the sigmoid.

C.—FORM AND CAUSE OF THE STENOSIS.

Gradual obstruction of the lumen of the bowel is the result of concentric narrowing, through disease of the intestinal wall, or of external pressure.

(1) *Concentric narrowing* occurs especially in cancer, tubercle, syphilis of the bowel, which is much rarer, and finally in non-specific cicatricial stenosis.

The differential diagnosis between **cancer** and **tubercle** is determined by the *age* of the patient and by the *localization* of the stenosis.

Tubercle is much more frequent than cancer in the small intestine, and is the only condition to be thought of, under the age of 20. After 30, the question of cancer must also be taken into consideration. Both diseases frequently occur in the ileo-cæcal valve and in the first portion of the cæcum; but cancer does not occur until the end of the third decade. The lower down the large intestine we proceed, the rarer does tubercle become and the more frequent is the incidence of malignant stenosis. The age incidence of cancer of the ascending and transverse colon is however younger than that of cancer of the sigmoid.

The *condition found on palpation* also has its significance. If a chronic obstruction in the small intestine is distinctly palpable, it is most likely to be carcinoma, because a tubercular stricture very easily escapes the palpating finger. But both carcinoma and tubercle are equally easily palpable over the ileo-cæcal valve. The latter is less sharply circumscribed, less hard, and less movable than the former.

In cases of tubercle, intestinal symptoms will already have been present for two or more years, periods of aggravation alternating with occasional longer periods of improvement. In cases of cancer, the patient will previously have enjoyed good health, but from the moment symptoms appear they continue to become progressively worse. There may be remissions, and occasional improvement in the general condition, but of very short duration, and despite the brief time during which the malady has existed, the emaciation is very striking. Exceptionally, cancerous stenosis may drag on for years before a definite clinical diagnosis can be made. If X-rays are not decisive, an exploratory laparotomy is preferable to indefinite waiting. It should also be mentioned that in cases of ileo-cæcal tuberculosis, a mixed infection often causes acute attacks of peri-typhlitis, which are at first mistaken for ordinary appendicitis.

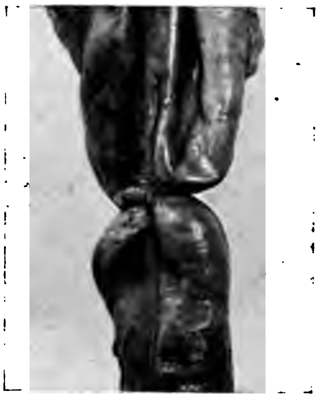


FIG. 193.—Cancer of the sigmoid in the shape of a constricting ring.

Carcinomata in the rest of the large intestine as far as the splenic flexure, are easily felt except in very fat persons, especially as they usually give rise to definite tumours, early in their course. The splenic flexure itself is not easily accessible to palpation, and it is necessary to get the patient gradually accustomed to the deep palpation which is required. The same applies to the hepatic flexure, especially in men. Cancer of the sigmoid cannot often be felt, because this part of the bowel so frequently lies in the true pelvis, and because the growth is usually very small. The bowel looks as if it has been tied round with a piece of string (fig. 193), and not as if it is affected by a growth. Bi-manual examination, under an anæsthetic, per rectum and abdominally, is often indispensable. If the obstruction is not found higher up, recto-sigmoidoscopy must not be neglected, because this is the only method which reveals the condition of the lowest 10 to 12 inches of the large intestine.

Cancer of the rectum, even if situated low down, may cause extreme narrowing of its lumen and produce symptoms resembling ileus. But the history of these cases shows that the chief complaint refers to tenesmus, so that to mistake a rectal cancer for one higher up is hardly conceivable, if a history is at all obtainable, and if a rectal examination has been performed, which ought never to be omitted in any case of obstruction.

Pure cicatricial constrictions are much rarer than the cancerous or tubercular variety. They should only be thought of when the history definitely suggests the possibility. Although strictures produced by typhoid and dysenteric ulcers do not possess the importance previously ascribed to them, we do know now that *injuries* of various kinds may lead to stenosis. For instance, *contusion of the bowel* may cause an infiltration in its wall, and this may lead to a temporary disturbance in its lumen, but it very rarely results in a permanent or in an increasing stenosis. The *tearing off of a piece of mesentery*, or what amounts to the same thing, *thrombosis of the mesenteric vessels*, is a much more serious matter. The interference with the circulation injures the mucous membrane, and by its destruction leads to a cicatricial stricture, even if the nutritive conditions are adequate for the other layers of the intestine.

A circular stenosis more often follows the *replacement of a strangulated hernia*, whether performed by the bloodless or open method. We will discuss this incident in connection with strangulated hernia.

For the sake of completeness we must mention *syphilitic* strictures, which, however, are always situated in the rectum and therefore cannot be confused with strictures higher up.

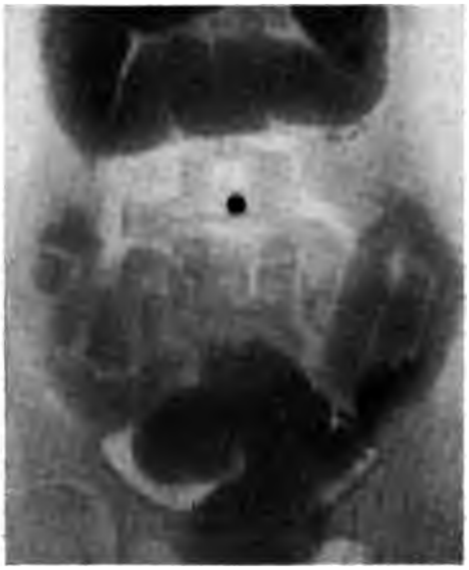


FIG. 194.—Diverticulosis of sigmoid (after bismuth injection.)

Sarcomata and innocent tumours (lipoma, myoma, polypus) rarely cause obstruction of gradual onset, but they may cause more or less complete obstruction through volvulus or intussusception.

Finally, it is necessary to mention stenosis of the lower portion of the large intestine, owing to chronic sigmoiditis or peri-sigmoiditis in the presence of Graser's diverticuli (diverticulosis). The diagnosis of this condition has hardly ever been made before operation.

But that it can be diagnosed is evident from the following case.

The patient, aged 65, had indefinite symptoms of obstruction of the bowels. Systematic examination—including the rectoscope—revealed nothing; but a skiagram, after a bismuth injection, supplied the key to the clinicial symptoms, and the operation confirmed the diagnosis of partial obstruction due to "sigmoiditis diverticularis" (fig. 194).

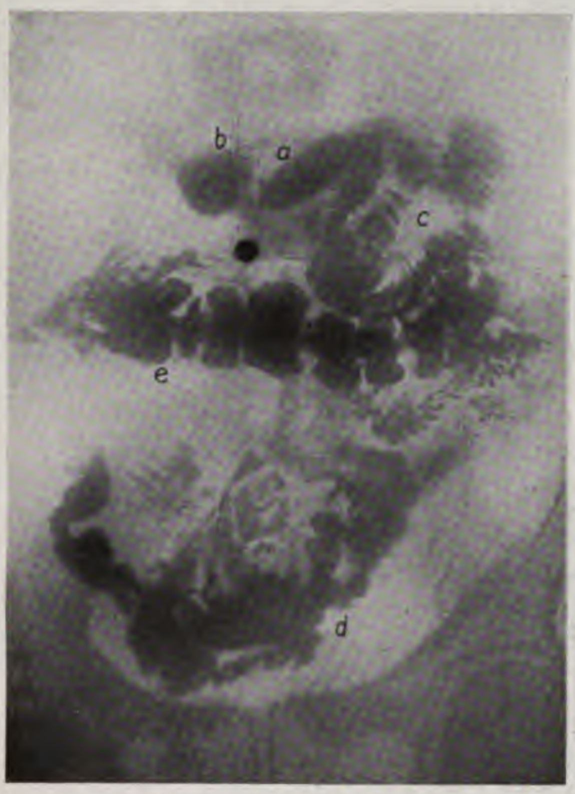


FIG. 195.—A bird's-eye view of the various intestinal appearances in a skiagram. (Case of cæcal tuberculosis six hours after a bismuth meal. *a* = Residue in stomach; *b* = bend of duodenum; *c* = jejunum with circular folds visible; *d* = lower portion of small intestine; *e* = transverse colon).

(2) We now turn to chronic ileus caused by *tumours pressing on the bowel from the outside.* The actual obstruction may depend upon one of three conditions—(1) on direct compression, (2) fixation of the bowel by adhesions, (3) infiltration into the wall of the bowel. The first condition is the rarest. As long as the bowel remains movable it can usually find some position in which its function can be maintained. Therefore it is that chronic ileus so rarely occurs in the most extensive innocent tumours as long as they are not affected

by inflammatory irritation. Even in cases where there is not much room for dilatation of the bowel away from the tumour—as in a fibro-myoma fixed in the true pelvis—the tumour and intestine do not usually interfere with one another. A **retroflexed pregnant uterus** is an exception to this rule, because of its unrestrained growth. But in the case of a malignant tumour which fixes itself to the adjacent bowel and prevents its dilatation, chronic obstruction from the pressure is very likely to occur, even if the growth has a comparatively small circumference. This occurs in **carcinoma** of the *kidney* and ovary, in large *cancers of the uterus,* and in sarcomata in various positions of the abdomen.

The symptoms of chronic obstruction in **inflammatory processes** are milder and always more transitory. In these cases the bowel is simultaneously compressed, fixed and infiltrated, the latter condition interfering to some extent with its normal peristalsis. This variety of obstruction is most prevalent in connection with abscesses and band formation after *peri-metritis, appendicitis,* and *peri-nephritis.*

Hitherto we have been assuming that chronic obstruction is, despite brief interruptions a slowly progressive malady, wherein the leisurely development of symptoms leaves adequate time for examination and reflection, but wherein the symptoms never cease completely. This is true for most cases, at least in a certain stage, but not for all cases, and especially not for the *beginning* of the disease. Chronic ileus has a tendency to be **intermittent**, although the cause of the disease continues unchanged. This depends upon the compensatory state of the intestinal musculature and possibly upon destructive changes within the stricture.

The less significant the symptoms are during a quiet interval, the more weight must be attached to the history. If the unequivocal signs of a stenosis have only once been reliably observed, i.e., localized colic, probably vomiting, circumscribed hardening of the bowel and the characteristic auscultation sounds, the case must be considered serious, even if all symptoms have temporarily disappeared.

Chronic obstruction is not always progressive. If it is caused by a moderate amount of scar tissue, e.g., after a strangulated hernia or an injury, it is quite possible that the symptoms may gradually abate and finally disappear completely. This is even more likely to occur in cases wherein the lumen of the bowel is interfered with by inflammatory processes. These cases constitute a fair proportion of instances of intestinal obstruction which recover without operation, in addition to cases of volvulus and intussusception to be discussed later on.

Much has been expected from *X-ray examination* in the diagnosis of intestinal obstruction, but not all the anticipations have been realized hitherto. The basis of the examination is a bismuth meal, just as in the case of the functional derangements of the large intestine, discussed in the previous chapter.

Narrowing of the *small intestine* is usually the result of tubercular stenosis. The narrowing is recognized by the stagnation of the bismuth meal in front of the stenosis. The small intestine should normally be quite empty four to six hours after a bismuth meal of 200 grm. of porridge, at least if this is not soon followed by another larger ordinary meal, which would tend to delay the expulsion of the bismuth meal from the stomach. As the contents of the small intestine are liquid, and its peristalsis is active, it follows that the stenosis must be very narrow, to cause any evident stasis of the bismuth meal.

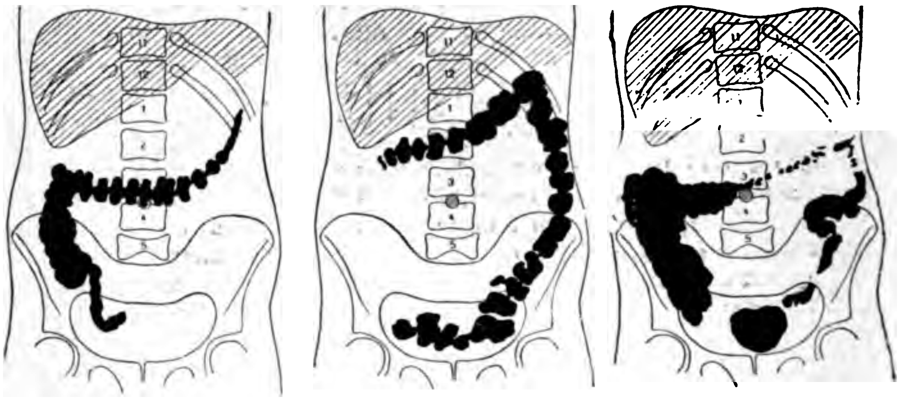
The following facts must be taken into consideration in regard to the large intestine :—

(*a*) The higher the position of the stenosis, and therefore the more liquid the fæces are, the narrower must be the stenosis in order to render it visible on the skiagram.

(*b*) If the stenosis is situated very low down, the stagnation does not occur just in front of it, but in the cæcum, as we have just seen. Constrictions of the descending colon and sigmoid are mainly indicated by an abnormally filled cæcum and ascending colon, this fullness persisting longer than usual. The same picture is also seen in constipation of the ascending type. We should, therefore, not suspect or assume the presence of an anatomical narrowing unless repeated skiagrams show an interruption in the fæcal column lower down in the large intestine, and always in the same place. Our suspicion will be confirmed if the fæcal masses always preserve the same shape at this spot. There may be a funnel-shaped gap in the shadow, indicating a narrow track in the bowel, or the loss of shadow may be at the side, just as in cancer of the stomach. Repeated examinations are indispensable, because such pictures may be the result of pure accident. In doubtful cases this test may be controlled by *injecting* 80 to 100 grm. of bismuth carbonate, or double this amount of barium sulphate, in 1½ or even 2 pints of very thin mucilage of starch. But as such a mucilage trickles through most constrictions, they are not always clearly demonstrated by this method unless a considerable loss of shadow occurs (fig. 196 *k*). Attempts have therefore been made to follow the injection on the screen, and the results have been better. The temporary stagnation of the material injected has rendered it possible to localize the stenosis in a few cases. (Haenisch.)

It is useful to take another skiagram after the spontaneous evacuation of the injection, for some stenoses are demonstrated better, when the plate is taken in this way, than when the bowel is full. But care must be exercised in the interpretation of the plate, because a simple kink due to adhesions may give the impression of a stenosis.

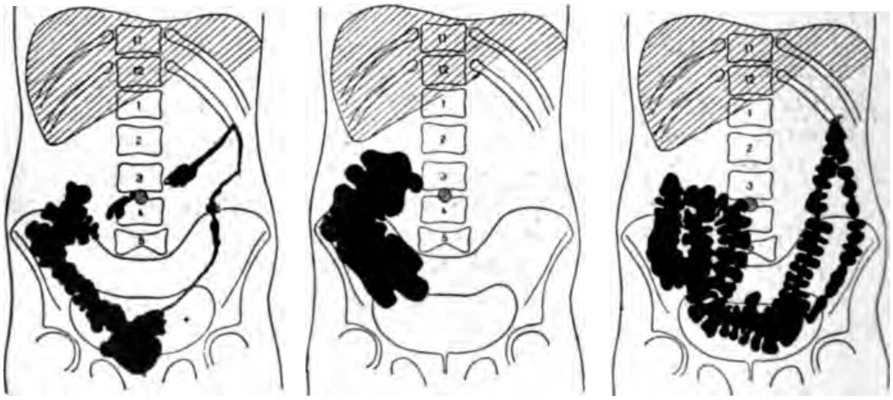
Skiagraphy renders great assistance in the diagnosis of intestinal disorder, and may be able to localize the disease when other methods fail, but as we have already said in connection with the stomach, it ought only to be employed for purposes of diagnosis in conjunction with other clinical aids.



(*a*)
Normal large intestine six to eight hours after bismuth meal.

(*b*)
Normal large intestine twenty to twenty-four hours after bismuth meal.

(*c*)
Large intestine after twenty-four hours in a case of spastic contraction of transverse colon.



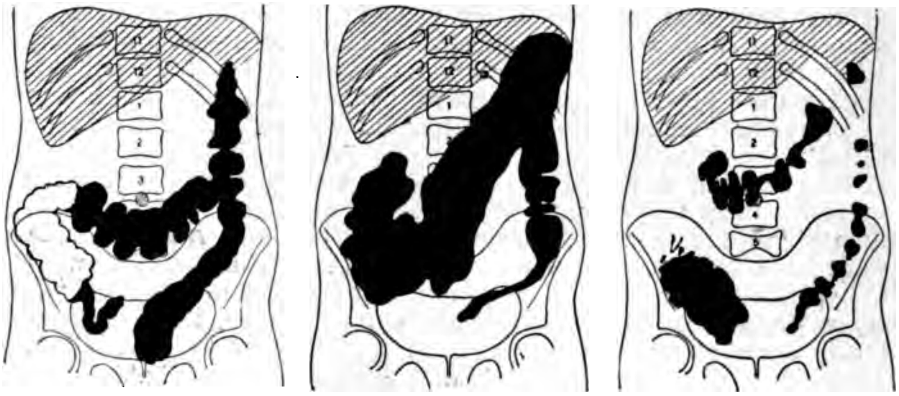
(*d*)
Severe degree of spastic colitis of lower portion of large intestine.

(*e*)
Ascending type of constipation (after forty-eight hours).

(*f*)
Diffuse form of constipation (after thirty hours).

FIG. 196.—Semi-diagrammatic illustrations of skiagraphy of large intestine.

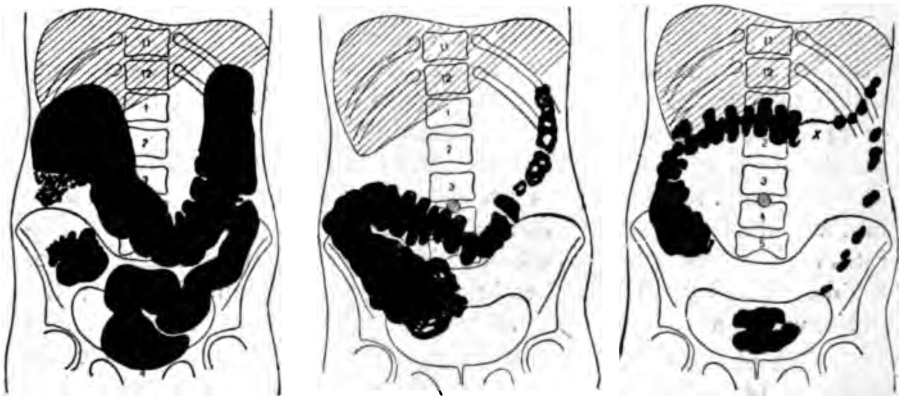
Hirschsprung's disease occupies a special position in the study of intestinal obstruction. The disease occurs in children—mostly in little boys—and is characterized by slight symptoms of obstruction



(*g*)
Large intestine in case of tubercle of cæcum and ascending colon. (Absence of shadow in diseased areas.)

(*h*)
Great tubercular infiltration of the sigmoid. Taken after bismuth injection. The diseased portion of bowel not thrown into folds.

(*i*)
Cancer of upper portion of ascending colon (after twenty-four hours). Circumscribed absence of shadow with proximal stasis.



(*k*)
Same case after injection. The diseased portion of the bowel does not become filled, although the healthy cæcum beyond does.

(*l*)
Stagnation of fæces in cæcum and ascending colon in a case of cancer of sigmoid.

(*m*)
Loss of shadow in cancer of transverse colon (x). Stagnation of fæces in cæcum. (Taken after twenty-four hours.

FIG. 196.—Semi-diagrammatic illustrations of skiagraphy of large intestine.

combined with extreme distension of the large intestine by fæcal masses. The blocked bowel is quite easily visible through the skin of the emaciated patient. On making a rectal examination masses of clay-like fæces are at once encountered, not only filling the ampulla but dilating it very considerably. The cleaning out of the bowel with the finger, spoons, and similar means, may occupy many hours.

Apart from the exceptional cases wherein the large bowel is unusually long and convoluted, or wherein the valve formation is abnormal, this disease is not due to anatomical changes, but merely to a purely functional disorder of defæcation. The little patients neglect their bowels either because of some accidental pain—fissure of anus—or because of some reflex disturbance, which would be called "sluggishness" in older patients. The habit of constipation is thus formed, if this may justly be termed a habit. Unless the mother notices this condition, the fæces collect first in the rectum, then fill up the sigmoid and finally extend beyond. After a certain stage has been reached, spontaneous evacuation is impossible, owing to hyper-distension of the bowel and sometimes also to some secondary valve mechanism. If assistance is not rendered, these children finally succumb to marasmus or symptoms similar to obstruction. The widely dilated coils of intestine are easily recognizable in a skiagram, with or without a bismuth meal.

II.—ACUTE INTESTINAL OBSTRUCTION.

A.—SYMPTOMS.

Acute intestinal obstruction differs from the chronic form in the suddenness of its onset and the completeness of the stoppage. It manifests, within the course of a few hours or of a day, the incidents which take weeks or months to develop in chronic cases, and shows more besides. In addition to the essential symptoms of intermittent colicky pains and localized contraction of the bowel, there occurs the important and regular sign of *vomiting*, which in chronic cases is only met with during an acute exacerbation. The general condition deteriorates rapidly owing to the lack of fluid intake and to the absorption of toxins. The urine diminishes in amount and contains indican; the pulse, which at first is quiet and full, soon becomes rapid and small; the breathing, which at first is only hurried during the actual colic, becomes rapid and shallow as meteorism increases, and the patient dies in a few days of hunger and thirst, unless peritonitis has ended the scene more quickly.

The diagnosis is often rendered difficult, because the symptoms of obstruction may be masked by those of the initial shock, which manifests itself by accelerated pulse and collapse. In a very severe case these symptoms merge almost uninterruptedly into the paralytic manifestations of the terminal stage, so that the pure signs of intestinal obstruction are not observed at all. In such a case the diagnosis may rest between acute perforative peritonitis and obstruction.

Perforation of a *gastric or duodenal ulcer*, with its disastrous symptoms, must be thought of in this connection, as also any condition which causes sudden shock and reflex intestinal paralysis, e.g., *pancreatic hæmorrhage and inflammation, torsion of an ovarian or omental tumour, embolism of the mesenteric arteries, tubal abortion, or ruptured tube, and even tabetic crises.* Repeated percussion and auscultation afford the best aid to diagnosis. If we repeatedly hear at any one place a metallic note, splashing or ringing noises, or, exceptionally, a stenotic murmur, and if the abdomen appears to be asymmetrical, with a localized area of the intestine, despite its tympanitic note, more resistant than its surroundings, we ought especially to think of ileus. But, on the other hand, the prevalence of dead silence from the beginning in an equally distended bowel most probably points to peritonitis, or at any rate to a severe toxic intestinal paralysis.

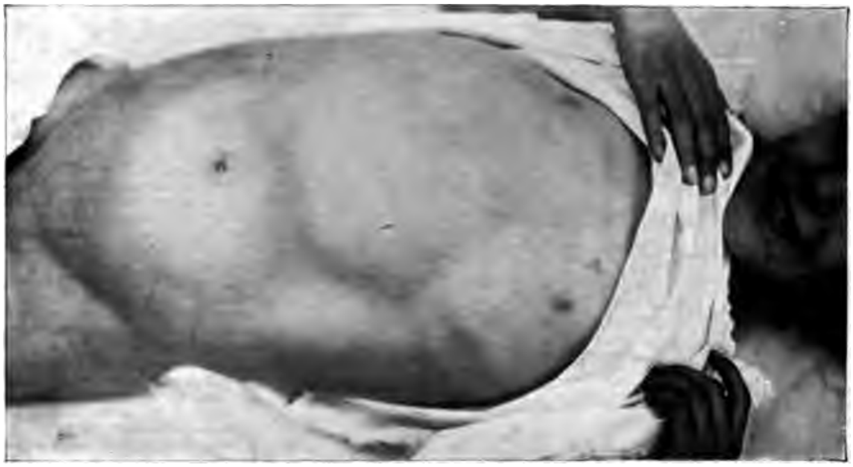


FIG. 197.—Hirschsprung's disease.

B.—THE POSITION OF THE OBSTRUCTION.

The diagnosis of the seat of obstruction is comparatively easy when it is either high or low; but difficult or impossible when it is in the mid-portions of the intestine. The most important indications are given in the accompanying table, and it is only necessary to add a few general observations.

The peristaltic movements in the small intestine are much more active than in the large intestine. But no conclusion can be drawn from the degree of meteorism, because it may be just as pronounced when the obstruction is low down in the small intestine, as when situated in the large bowel. "Meteorism in the flanks" ('cadre colique'), ostensibly a sign of obstruction in the large intestine, is

(*a*) *Obstruction at cardia.*—Abdomen flat, regurgitation of food by cupfuls, mixed with blood and mucus, often alternating with vomiting. Cancer, rarely cardio-spasm.

(*b*) *Pyloric obstruction.*—Epigastrium distended, rest of abdomen flat. Vomiting, by the dishful, of food taken days before, mixed with gastric juice and often with mucus, blood and coffee grounds. No bile. Stenosis after ulcer. Cancer.

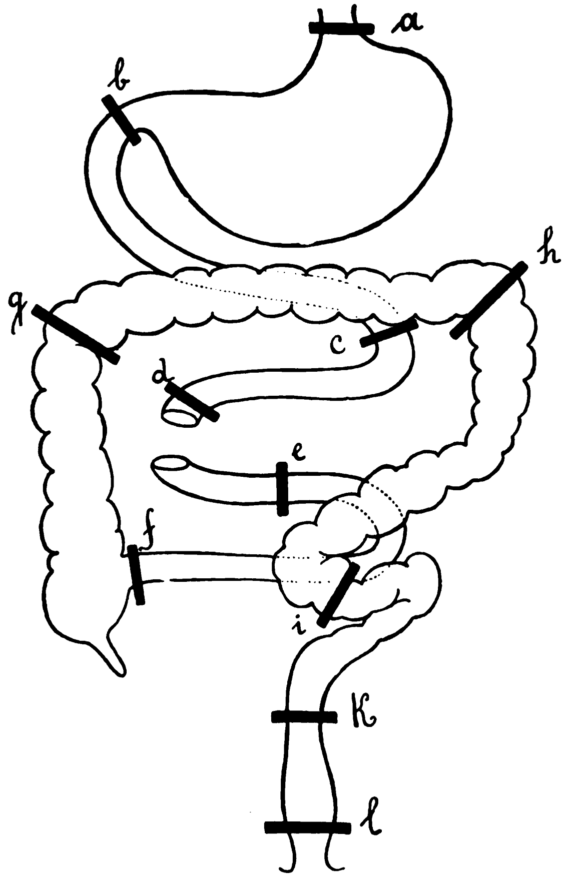


FIG. 198.—Diagram of the typical positions of intestinal obstruction.

(*c*) *Duodeno-jejunal obstruction.*—Abdomen as in *b*. Splashing sounds to the right of umbilicus (lower part of duodenum). Biliary vomiting, not fæcal. Arterio-mesenteric intestinal obstruction, Treitz's hernia, tubercular band.

(*d*) *Obstruction at upper part of small intestine.* — Meteorism, if present, moderate, central or diffuse. Vomiting biliary, rather fæcal, powerful peristalsis. Bands, volvulus, tubercle, tumours, intussusception, internal hernia.

(*e*) *Obstruction at lower part of small intestine.*—Meteorism, if present, general. Fæcal vomiting, powerful peristalsis. (Causes as in *d*.)

(*f*) *Obstruction at ileo-cæcal valve.* — As in *e*, but condition usually palpable in right pelvic cavity. Intussusception, volvulus, cancer, tubercle.

(*g*) *Obstruction of large intestine at hepatic flexure.*—Meteorism if present, general. Cæcum and ascending colon distended. Active peristalsis occasionally. Fæcal vomiting. Possible to inject 1½ to 2 litres of fluid into rectum. If obstruction incomplete, stools are formed. Condition generally palpable. Cancer, tubercle very rarely.

(*h*) *Obstruction of large intestine at splenic flexure.*—Meteorism as above, but transverse colon also somewhat distended. Injections of 1 to 1½ litres possible. If obstruction incomplete, stools generally formed. Palpation more often negative than in *g*. Cancer-tubercle very rarely, or syphilis.

(*i*) *Obstruction at sigmoid.*—Meteorism as above, main distension of colon at cæcum. Slight peristalsis. Injections of ¼ to ½ litre possible, seldom more. If obstruction incomplete, diarrhœa alternates with constipation. Palpation often negative owing to smallness of growth, but sigmoidoscopy and bi-manual examination under chloroform advisable. Volvulus, cancer.

(*k*) *Obstruction in upper part of rectum.*—Meteorism as above. Constipation alternating with diarrhœa, or always thin evacuations. Exceptionally ribbon, shaped. Tenesmus occasionally. Cause detected by rectal or combined examination. Cancer, syphilis, tumours and inflammation within true pelvis.

(*l*) *Obstruction in ampulla.*—Tenesmus, fluid or ribbon-shaped stools. Causes: Cancer, syphilis. Can be felt and sometimes seen.

more of a theoretical condition, because only the upper and middle portions of the bowel distend to any great extent—from the cæcum to the transverse colon—so that the frame is only half formed. If the distension proceeds further onwards, and the sigmoid is well developed, it will involve the mid-portion of the lower abdomen, but will not form a frame. But we may consider it established that a high tympanitic note in the right lumbar region points with great probability to obstruction of the large bowel.

Obstruction high up leads to many mistakes in diagnosis, because flatus and stools still pass and the abdomen remains flat, even if the obstruction is long persistent. These cases often suggest cerebral, uræmic or even hysterical vomiting, gastric crises or the onset of peritonitis. The last, however, can be excluded by the absence of any local irritative symptoms, uræmia by the condition of the urine, cerebral or tabetic vomiting by the absence of any other cerebral or spinal cord symptom. The obstinacy of the vomiting, the rapid decrease of urine and the cessation of the passage of stools and flatus within a few days will finally convince those who cannot conceive of intestinal obstruction without a drum-like abdomen.

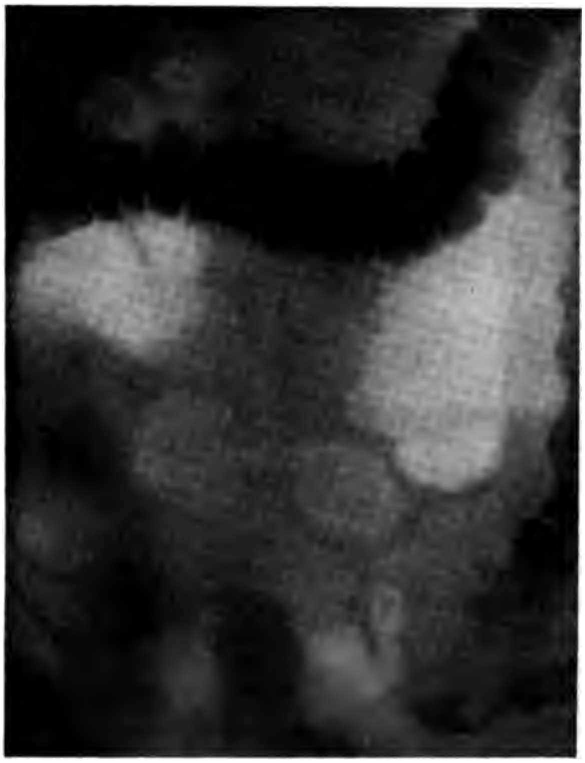


FIG. 199.—**Obstruction of small intestine. The injection indicates patency of large intestine. Great distension of small intestine.**

We cannot expect much elucidation from X-rays in cases of complete intestinal obstruction. There is no time for an examination of the intestinal movements, apart from the fact that a bismuth meal is liable to be vomited. Even if it were retained, the site of the stagnation of the test material is not the position of the obstruction. In obstruction of the large intestine, its position may, however, be shown on a skiagram, after a bismuth injection.

C.—THE GENERAL VARIETIES OF ACUTE OBSTRUCTION.

Before discussing the detailed causes of acute obstruction, we will glance at the different forms in which it may appear. This consideration will often facilitate accurate diagnosis. The following are the main varieties :—

(1) *Obstruction passing from an Incomplete Chronic into a Temporarily Complete—apparently Acute, Stage.*—Cancerous and tubercular disease constitute the chief examples of this group. If an elderly patient who has been suffering for several months from colic and increasing distension, has passed neither stools nor flatus for two days, but vomits bowls full of brown fœtid material, it is highly probable that he has a constricting carcinoma of the colon, particularly of the sigmoid. If a younger man with tubercular antecedents and a history of months' or years' suffering from colic, shows signs of complete obstruction, he probably has a tubercular structure in the small intestine or at Bauhin's valve. Many cases of obstruction during the course of tubercle or cancer of the peritoneum should be included in this group. In both, the apparently sudden onset of complete obstruction will always have been preceded by some abdominal discomfort, especially colic and loss of appetite, which shows that the event has long been in preparation.

(2) *Intermittent Obstruction.*—This term applies to all cases wherein sudden attacks of transitory obstruction alternate with intervals of complete freedom of a variable duration, sometimes even of years. There is no permanent narrowing in these cases, but a temporary obstruction of the lumen of the bowel repeatedly occurs, caused by some existing abnormality, which becomes latent at intervals, or by some anatomical change. This variety is mostly exemplified in torsion of the sigmoid, or more rarely by an abnormally movable ileo-cæcal segment of the bowel, or, still more rarely, of the small intestine. Obstruction by omental or cicatricial bands, by abnormalities connected with Meckel's diverticulum, strangulation of internal hernia, kinking produced by tubercular adhesions, as well as arterio-mesenteric intestinal obstruction, may belong to the same group.

(3) *Sudden and unanticipated Onset of Acute Obstruction.*—This

group includes the rare cases of sudden obstruction by cancer in apparently healthy individuals without any pathological antecedents. But the more carefully the history is taken, the more frequently some indication of previous disease will be found, if only some dyspepsia, slight pain, irregularity of the bowels, or unexplained wasting. Obstruction may also occur suddenly in tubercle, before any diagnosis of intestinal disease has been made.

All the enumerated causes of intermittent obstruction come into consideration again if a first attack is under observation. Finally, there are cases in which, as a rule, one attack only occurs, e.g., intestinal obstruction from gall-stones.

D.—CAUSES OF ACUTE INTESTINAL OBSTRUCTION.

We now propose to consider whether any given case can be referred to one of the classical forms on the evidence of history and physical symptoms, and for this purpose we will briefly discuss the most important of these forms, beginning with those to which the history provides the clearest clues.

(1) Obstruction due to Bands and Kinks.

If the patient has had an abdominal operation, however long ago, we shall rarely err if we diagnose obstruction by a band. Bands, which result from operative procedures are more dangerous, because they are usually more circumscribed than those which arise spontaneously after inflammation. Nevertheless, the latter may also produce obstruction by bands, e.g., after appendicitis, cholecystitis, inflammation of the female genital organs, and tubercular peritonitis. The last may produce adherent omental bands in the true pelvis. The adhesions, which occur after ulceration of the large intestine, pericolic cords and bands, are flatter, and therefore are more liable to give rise to chronic obstruction.

If signs of obstruction in the upper part of the small intestine occur in an emaciated individual, the subject of lateral curvature or Pott's disease, we should think of *arterio-mesenteric intestinal obstruction*, i.e., kinking of the small intestine at the duodenal boundary by the root of the mesentery. A duodenum with a very low-lying situation is necessary, or at any rate is a favourable condition for the causation of this form of intestinal obstruction. The knee-elbow position removes the obstruction and confirms the diagnosis.

The classical picture of arterio-mesenteric intestinal obstruction was seen in a girl, aged 13, the subject of disease of the cervical spine and compensatory lordosis in the lumbar region. The stomach and the duodenum, which, as the operation showed, reached considerably to the right, were greatly distended, and gave evidence of splashing on auscultation. The stomach was tympanitic and the duodenum yielded a metallic note. The cervical disease prevented the use of the knee-elbow position for the purpose of treatment, and the fact of tuberculosis did not, of course, exclude obstruction by a band. Laparotomy was, therefore, performed, and I found the whole of the small intestine deeply in the true pelvis. The kink was situated at the junction of the duodenum with the jejunum. The latter became filled with gas as soon as it was lifted up.

It is an open question whether one should include here the duodenal obstruction which has been observed after laparotomy, especially in the upper portion of the abdomen. This condition depends upon atony of the gastric musculature, which sometimes occurs after operations on the abdominal cavity, and is analogous to atony of the rectum. It is a condition which certainly occurs in the slight infections which are overcome by the peritoneum in a few days. In other cases the peritoneum possesses an idiosyncrasy towards blood.

(2) Obstruction by Gall-stones.

If pain on pressure over the gall-bladder occurs *simultaneously* with symptoms of acute intestinal obstruction, the case is probably one of acute cholecystitis. This disease frequently produces temporary signs of obstruction, either as a reflex process or by extension of the inflammation to the transverse colon or small intestine.

If an attack of gall-stones *has occurred previously* the obstruction is most likely to be due to a **gall-stone.** The entrance, even of a large gall-stone into the small intestine, usually takes place by a process of suppuration without any striking symptoms, and the last attack of gall-stones observed by the patient may have occurred long previously. Some support for the diagnosis of "gall-stone obstruction" is afforded by feeling a firm and somewhat tender swelling in Douglas' pouch on vaginal or rectal examination. Most of these gall-stones remain fixed in the lower portion of the small intestine, and the coil containing the stone is dragged down into the cavity of the true pelvis.

It might be thought that gall-stone obstruction ought to be included in the chronic variety, owing to the length of time the stone remains within the bowel. This is true for some cases, wherein the symptoms are very protracted and remittent, and the stone eventually departs spontaneously. But in most cases the symptoms come on very acutely, and we often find that signs of peritonitis supervene at the same time, or at any rate very soon afterwards. If the stone is in a healthy coil of intestine, it will allow flatus to pass by it. But if it remains for any length of time, the intestinal wall becomes irritated and draws itself tightly over the stone, which then allows nothing to pass by it. At the same time a pressure ulcer develops, so that the intestinal wall becomes inflamed and infiltrated, and instead of grasping it by active contraction, becomes stiff by infiltration, and embraces the stone tightly but passively. At this moment the "attack of gall-stone obstruction" often begins. The inflammation rapidly involves the serous membrane, so that local peritonitis occurs early. If we make the diagnosis of gall-stone obstruction in such a case on the evidence of the clinical history, we must not hesitate to operate *immediately*, on the assumption that most gall-stones pass through the bowel by themselves. The actual symptoms, indeed, show that the stone, in such a case, will not do so.

The following is an illustrative case :—

A man, aged about 50, who had suffered from a severe attack of gall-stones one year previously, was suddenly seized at night, without any warning, with abdominal pain and vomiting. Twelve hours later, the pulse was already rapid, the abdomen tender and slightly distended, and contained some free fluid. The vomiting continued. The diagnosis wavered between obstruction by a band, or by a gall-stone, combined with severe peritoneal irritation and slight effusion. Operation was done at once, and a gall-stone was found in the lower portion of the small intestine, in firm contact with the inflamed and infiltrated bowel. A pressure ulcer had already perforated, and the abdomen contained sero-purulent fluid. The operation, however, was unable to restrain the advance of the peritonitis.

(3) Intussusception.

Exceptionally, a positive indication is supplied by the age of the patient. This is true of **intussusception** in so far as it occurs in infants, who very rarely suffer from any other form of obstruction. The symptoms vary with the degree of disturbance of the circulation in the invaginated portion, and they range from the very severest type, with rapid death from peritonitis, to chronic intussusception of many months' duration. The beginning of the disease is usually marked by the passage of blood-stained, fruit-juice-like fluid with the stools, and the intussusception can, as a rule, be felt, on careful examination, as a sausage-shaped swelling, close to the spinal column. The intussusception rarely reaches so far as to be felt in the rectum, like the vaginal portion of the cervix, or be mistaken for a prolapse of the rectum and operated on.

Intussusceptions which are not of the ileo-cæcal variety are as a rule, caused by *Meckel's diverticulum* or *innocent tumours of the bowel.* In both cases the upper portion of the intestine with the tumour, or with the inverted diverticulum, is dragged into the lower portion. I once saw the characteristic etiology well exemplified in a little boy; the lower portion of intestine was fixed by tubercular mesenteric glands, the upper, forming the intussusception, was, however, free. This corresponds to the condition at the ileo-cæcal valve, where the more movable portion of the intestine becomes inverted into a less movable portion.

We have mentioned the passage of blood-stained liquid as an important sign of intussusception. The same symptom occurs in *infarction* of the bowel, caused by blocking of a blood-vessel, which is usually accompanied by the symptoms of sudden intestinal obstruction. The simultaneous *vomiting* of blood-stained material points to infarction.

(4) Volvulus.

There is a geographical indication which points to **volvulus** as the diagnosis of the form of intestinal obstruction. In most countries, neither the student nor surgeon sees many instances of this condition, but in Russia, especially in the Baltic Provinces, volvulus is the most frequent variety of obstruction brought into hospital.

Gruber states that this is due to the greater length of the Russian intestine; others attribute it to the abundant dietary of potatoes. I know many places where the potato is a great favourite, but where volvulus is almost unknown. Gruber's view is also stoutly contested. Indeed, volvulus is more likely to be due to congenital or acquired abnormalities in the mesentery, which permit a more extensive movement of some parts of the intestine, than to the length of the bowel in metres. To allow of the twisting of the bowel the coil requires a certain amount of independence, which is given it by a long mesentery with a narrow attachment. Such a condition is only normal in the sigmoid, and it can be increased by the greater development of this coil or by the abnormal approximation of its lower extremity. Sometimes the ileo-cæcal portion is provided with so profuse a mesentery that it occasionally twists about on its long axis. Finally, the *whole of the small intestine*, either itself or with the *large intestine*, may possess such a narrowly attached mesentery that the whole bowel is capable of twisting in its entirety. I saw an instance of this in Kocher's wards, in a young man in whom the torsion occurred after an enormous meal of cherries, including the stones. The occurrence of torsion in a *single coil of small intestine*, presupposes a condition in which it has been subjected to traction for a considerable time, such as long detention in a hernial sac, or the pull of an intestinal tumour.

The diagnosis of volvulus of the sigmoid is very easy. In this condition the whole abdomen is occupied by an enormously distended coil, with its head in the upper part, and with its more or less parallel limbs, which can be distinctly felt, and even seen. Confirmation is afforded by the impermeability of the sigmoid to an injection of water. An *ileo-cæcal volvulus* may be diagnosed, if instead of a long and distended coil, we find that a roundish tympanitic tumour has suddenly formed, with a seizure of vomiting, and that an injection of water can be successfully given. Volvulus of the *whole small intestine* yields the symptoms of duodeno-jejunal obstruction, plus meteorism. The symptoms of volvulus of the *small and large intestine* together are about the same, but it is impossible to inject any large quantity of water into the rectum.

It is not possible to differentiate volvulus of a *single coil of small intestine* from obstruction by a band, and from strangulation into a peritoneal pouch.

It is equally impossible to detect clinically the *nodules* which are to be found in the vicinity of a volvulus, and which depend upon the presence of Meckel's diverticulum and the bands of connective tissue arising from it. Certain forms of obstruction due to congenital bands are also undiagnosable clinically.

The foregoing remarks apply to volvulus which has caused complete obstruction and a torsion of 360°. If the obstruction is incomplete (torsion of 180° to 270°), the symptoms are less severe, and often disappear spontaneously. For instance, I once saw the desired evacuation of flatus occur after a long journey, at the very moment when everything was about to be expedited for an operation. In all these cases, however, the diagnosis is only a matter of pure probability.

(5) Strangulation of Internal Herniæ.

Strangulation into a congenital peritoneal pouch is one of the rare causes of intestinal obstruction; but sometimes this diagnosis is suggested by the demonstration of a localized distension. But before we think of an internal hernia, we must exclude *strangulation of an external hernia*, which is not always so easy to do as it may appear, especially in cases of pro-peritoneal and inter-muscular inguinal herniæ—herniæ of the obturator foramen, lumbar, and gluteal herniæ.

I recall a case which occurred when I was an assistant. A young woman was sent into the surgical ward after suffering from obstruction for several days. She had already been examined for hernia, but none had been detected. Nevertheless a slight resistance at the apex of the internal inguinal ring pointed to strangulation of an interstitial hernia; and at the operation it was found that the contents of the hernia were already gangrenous at the constricting ring. Unfortunately this error is of frequent occurrence.

An error in connection with an obturator hernia is more pardonable. In a typical case, however, the pain or deep pressure below Poupart's ligament, and the neuralgia of the obturator nerve—probably called old rheumatism by the patient—allows the diagnosis to be made with the greatest probability.

But, on the other hand, not every hernia which may accidentally be present, even if irreducible, must be credited with the causation of intestinal obstruction. If the hernia is not tense, and if there is no pain on pressure over its neck, it has nothing to do with the obstruction, even if old adhesions have rendered it irreducible.

If a hernia has been reduced previously to the occurrence of obstruction, the neighbourhood of the ring must be examined. Any in-drawing of the tissues, or indefinite resistance, or pain on deep pressure points to *reduction en masse;* but if the hernial ring is normal, it indicates that the obstruction has some other origin.

There are three particularly important forms of **internal hernia**: (*a*) *Hernia* at the *Foramen of Winslow.* When strangulated, this causes a tumour behind the stomach. It has often been operated on, but has not previously been diagnosed. (*b*) Diagnosis ought to be more possible in *duodeno-jejunal hernia.*

This begins at Treitz's pouch, where the jejunum passes under the transverse colon. The pouch opens to the left of the first part of the jejunum, and is directed thence obliquely upwards and to the left. It may become large enough to contain the major portion, or indeed, the whole of the small intestine.

The symptoms of these herniæ are intermittent in character; but this circumstance is common to several other forms of intestinal obstruction. The hernial tumour is situated in the epigastrium, rather towards the left. There have been cases recorded wherein strangulation has occurred in a similar pouch arising from the right of the jejunum.

(*c*) The third typical position for internal herniæ is in the *appendix region.*

Of the various pouches described by anatomists only two possess surgical importance, viz., the *ileo-appendicular,* which is situated between the appendix and the end of the small intestine under the region of Bauhin's valve; and the *retro-cæcal,* which runs laterally from the appendix behind the cæcum.

In both forms a hernial swelling is found in the ileo-cæcal region, and if no other cause for the intestinal obstruction is evident, it is fair to assume that one of these varieties of hernia is responsible.

(*d*) **Diaphragmatic herniæ** are seldom suspected before operation, unless a previous injury to the diaphragm has suggested its probability. A tympanitic note or dulness over the left lower lobe and marked dysphagia might arouse suspicion. These herniæ are either on the left side, or in a space in the diaphragmatic attachment to both sides of the sternum (Morgagni's space). An X-ray examination after a bismuth meal furnishes the best means of diagnosis.

We shall not mention any of the rarer forms of internal hernia, for it is quite impossible to diagnose them clinically. This applies to strangulation in openings of the mesentery, omentum, or broad ligament.

(6) Spastic Obstruction.

Spastic contractions of the intestine, without any evident cause, sometimes occur after abdominal operations, and if they persist for any length of time lead to symptoms of intestinal obstruction, i.e., **spastic obstruction.** The following is a typical case:—

A young, healthy man came to the hospital with symptoms of obstruction high up in the small intestine and a temperature of 99·6° F. The family history suggested tubercle. It was remarkable that his general condition remained comparatively good, despite the continuance of the obstruction. Operation showed that a coil 15 cm. in length, in the upper part of the small intestine, had contracted to a thin band. When the spasm relaxed all symptoms vanished.

It is possible that atropine might have been effective here, and permitted the making of a diagnosis. As a rule, no diagnosis is made until the abdomen is opened; spastic conditions of the large intestine are more frequent, but less serious.

Persistent vomiting must not be confused with spastic obstruction, because hysteria may imitate intestinal obstruction. But in that condition the physical signs do not correspond with the general severity of the symptoms arranged by the patient. If it is pretended that solid fæces are vomited, this must baffle even those who easily believe what they are told, because lumps of fæces are not vomited by anti-peristalsis. They get into the vomit in a much simpler way. Relapses have been observed in cases wherein the first "exhibition" was successful. A thorough course of psychical treatment is required to prevent these relapses.

CHAPTER XLIX.

TUMOURS AND SWELLINGS IN THE ABDOMINAL PARIETES.

THE superficial situation of a tumour or swelling indicates that it is in the abdominal wall and not in the abdominal cavity. But in very thin people a genuine intra-abdominal tumour may appear to be quite superficial. It is, therefore, necessary to make the abdominal muscles contract. If the tumour then disappears, it must be situated within the abdominal cavity, or, at any rate, under the abdominal muscles. If however, the tumour can still be felt, and at the same time becomes immovable, it is obviously connected with the muscles or fasciæ. Should the muscular contraction make no difference at all, the tumour is situated either within the skin or subcutaneously.

In order to obtain a correct idea amid all the various possibilities, it is very important to note whether the tumour is in a position "typical" of these pathological changes, i.e., in the middle line, in the inguinal region, in the lumbar region, or whether it has appeared at some other point.

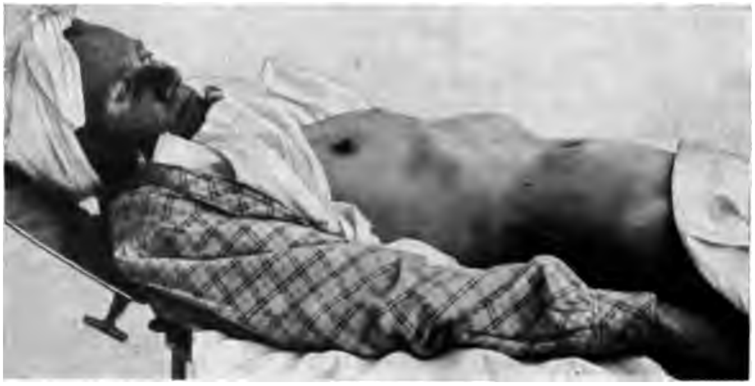


FIG. 200.—Chronic abscess in epigastrium (probably originating in a gastric ulcer).

(I) THE UPPER ABDOMINAL REGION.

A swelling in the epigastric region may apart from exceptional rarities, be one of three things; viz., (1) an epigastric abscess; (2) subcutaneous lipoma, or (3) an epigastric hernia.

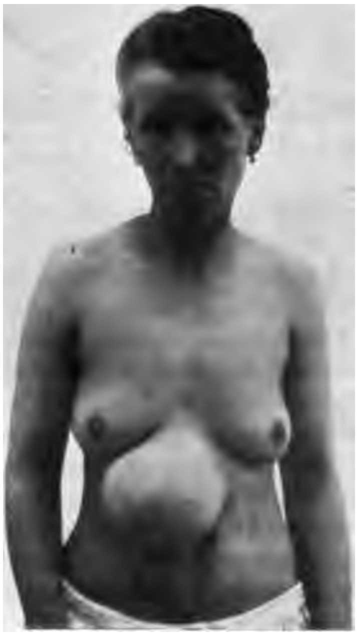


FIG. 201.—Subcutaneous lipoma of the epigastrium.

If the swelling is acute, and presents the appearance of a firm infiltration, which, after a little while, begins to soften in the centre and fluctuate, there is no difficulty in recognizing an **epigastric abscess.** The chronic form, in which the skin remains unchanged for a considerable time, may easily give rise to doubt. But even in these cases the wide base of the swelling (fig. 200), and the fluctuation which is rarely absent, suffice to indicate an abscess. The source of infection is often a gastric ulcer which has become adherent to the abdominal wall, and which has caused suppuration without direct perforation. In other cases, the swelling may be an abscess due to tubercle of the ribs or sternum. Rarely it may be a burrowing abscess making its way through the abdominal muscles.

Subcutaneous lipomata differ in no respect from lipomata in other situations. The accompanying illustration (fig. 201) shows that they may attain a very large size. They are distinguished from epigastric herniæ and from subserous lipomata, by their free mobility over the sheath of the rectus.

Epigastric herniæ and subserous lipomata are, however, much more frequent.

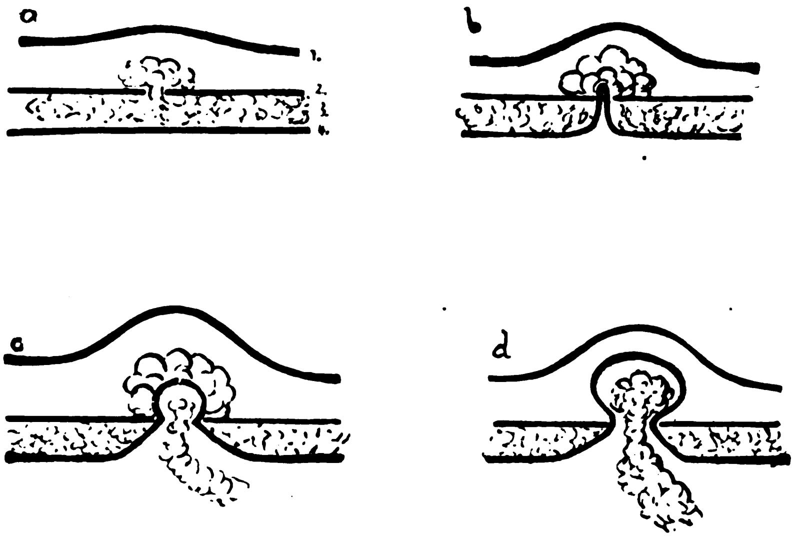


FIG. 202.—Epigastric hernia and subserous lipoma.

(1) Skin. (2) Rectus sheath. (3) Subserous fat. (4) Peritoneum.
(*a*) Subserous lipoma, after breaking through sheath of rectus.
(*b*) ,, ,, with some peritoneum pulled up into fascial slit.
(*c*) ,, ,, with a hernial sac containing some protruded omentum. (Epigastric atty hernia).
(*d*) Pure epigastric hernia, without lipoma.

In order to appreciate their origin we must realize that a large amount of fat is contained in the upper triangular area of peritoneum which has its apex at the umbilicus. The first stage in the development of a hernia is the protrusion of a lobule of fat through an oval slit, which is always situated transversely in the fibrous tissue of the linea alba. If this lobule continues to grow after it has become free, it develops into an ordinary *subserous lipoma* (fig. 202 *a*). As it grows it usually pulls up some peritoneum into the slit, so that it obtains a pedicle of peritoneum, which, however, contains no abdominal viscus within it (fig. 202 *b*). If this peritoneal protrusion in front of the slit in the aponeurosis develops into a hernial sac, into which omentum or bowel finds its way, the case is one of *epigastric fatty hernia* (fig. 202, *c*). If, finally, the hernial development is greater than the fatty proliferation, the case is one of ordinary epigastric hernia (fig. 202, *d*).

It is not possible to differentiate definitely between a true hernia with reducible omentum and a subserous lipoma, because subserous fat can be squeezed back through the slit in the linea alba. Irreducibility is, of course, in favour of a simple lipoma, but omentum may also become irreducible, owing to swelling and adhesions. These structures are usually small, as in fig. 203. Large epigastric herniæ, as in fig. 204, are rare.

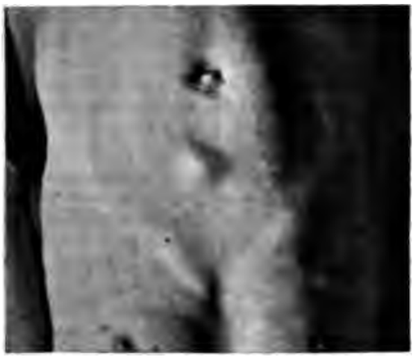


FIG. 203.—Small epigastric hernia.

If the diagnosis of epigastric hernia were always made in time, it would prevent many so-called "dyspeptics" wasting years on treatment for indigestion, when a simple surgical measure could at once restore them to health. The "dyspepsia" is caused by the pull on the peritoneum, or on the omentum or ligamentum teres of the liver, firmly grasped within the hernial sac. But, on the other hand, one must not overlook a cancer of the stomach, or duodenal ulcer, because there happens to be an epigastric hernia present. Both the abdominal wall and the stomach must be investigated.

(2) THE UMBILICAL REGION.

If we find that a newly-born infant has a swelling in the umbilical region, generally with a broad base, and but rarely pedunculated, in which the intestinal contents—usually the liver and bowel—can be seen through a veil-like membrane, the case is clearly one of **hernia of the umbilical cord** (fig. 206). It is quite impossible to mistake this for any other condition, and therefore need not detain us.

If the umbilicus projects in a semi-globular form and eventually in a conical or cylindrical shape, in a shrieking *little child*, only *one* diagnosis is possible, that of **umbilical hernia.**

If an adult, generally over forty, presents a tumour, varying in size from a pea to a man's head, in the same region, the same diagnosis applies, if the swelling is at least partially reducible. The principal diagnostic interest in these cases, is not so much concerned with the *nature* of the trouble, as with certain of its *accompanying symptoms.* If the contents are reducible, either the granular feel or the gurgling sound will indicate omentum or intestine. But in some umbilical herniæ it is impossible to demonstrate the one or the other. The contents are easily displaced by pressure, but neither the reduction nor the refilling is accomplished with a jerk, which suggests that the contents must be fluid. On careful examination, free fluid will also be found in the abdominal cavity, and the liver may exhibit signs of cirrhosis, or some cause for the ascites may be discovered in the heart or kidneys (fig. 205).

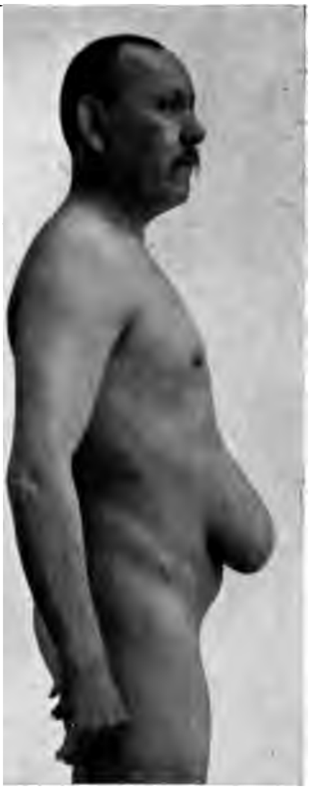


FIG. 204.—Large epigastric hernia.

In large old-standing cases of umbilical hernia (fig. 205) it can be seen, even externally, that they consist of separate loculi. Some of these may be reducible while others are not. It sometimes happens that a hard and tender swelling arises in one of these loculi, while the rest of the hernia still remains reducible. The most likely explanation of this occurrence is the strangulation of intestine or omentum in one of the loculi.

But other causes are conceivable. For instance, in the case of an old woman, whom I suspected of such partial strangulation, the localized inflammatory symptoms were really caused by a tubercular peritonitis which had involved the hernial sac. This might very well occur also with cancerous peritonitis. On another occasion I witnessed a peritonitis, which arose from the suppuration of an ovarian cyst, involve a large omental hernia and form a circumscribed abscess. An inflamed gall-bladder, and even the appendix has been found in an umbilical hernia.

If the tumour presenting at the umbilicus is not reducible at any stage we must ascertain whether it fluctuates, or, at any rate, whether it feels elastic, in which circumstance it may be one of the various cysts occurring in this region, especially a **dermoid** or epidermoid. If the cyst is situated in or beneath the abdominal wall, opposite the

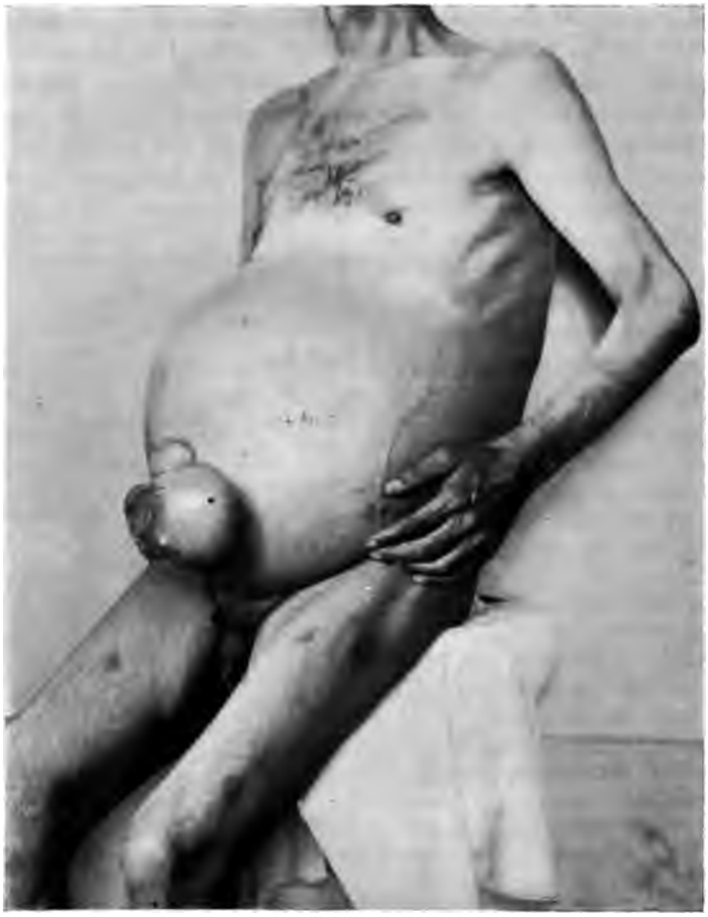


FIG. 205.—Multilocular umbilical hernia in cirrhosis of the liver.

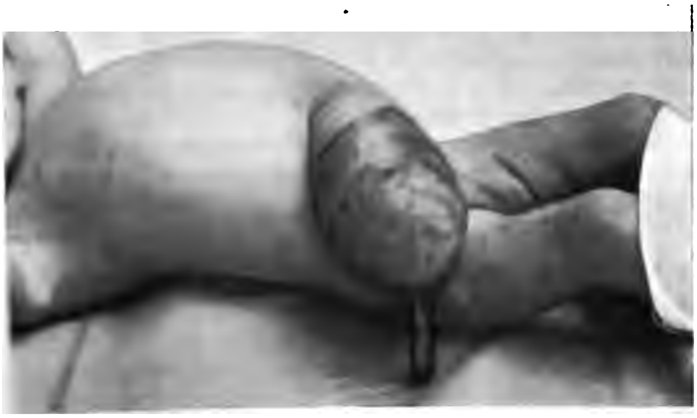


FIG. 206.—Hernia of the umbilical cord in an infant.

bladder, it is probably connected with the **urachus**; if directly behind the umbilicus, with the **vitelline duct.**

Solid tumours are usually **secondary** and represent metastases or direct extensions of cancer within the abdominal cavity. If, however, such a causation can be excluded, they must, apart from rare exceptions, be regarded as **primary cancers** or **sarcomata** if they grow rapidly, and as fibromata if their growth is slow.

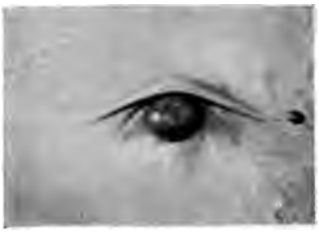


FIG. 207.—Cancer of the umbilicus in a case of carcinoma of the gall-bladder.

The primary cancers start from the *skin* and appear as ulcers with hard margin and base, or as cauliflower-like papillomata. Cancer of the umbilicus may also arise from *intestinal epithelium*, which has become displaced into the umbilical scar. Finally, the epithelium of the *urachus* may undergo cancerous degeneration. We should think of this origin, if the cancerous tumour stretches downward from the umbilicus towards the bladder. The *sub-umbilical abscess* may also be mentioned. This occupies a triangular area with its base upwards under the umbilicus and behind the recti. The pus organisms reach this space from the viscera of the lower abdominal cavity, or from the abdominal integuments. The course of this abscess is either chronic or acute, depending upon the nature of the organism—tubercle bacillus, staphylococcus, colon bacillus, &c.

(3) INGUINAL REGION.

Owing to the presence of the spermatic cord, or the round ligament, and the vaginal process of peritoneum, the inguinal region is the seat of many kinds of swelling, which we shall meet with later on. For the moment we are only concerned with the leading features of diagnosis in this region.

We must first ascertain whether the swelling can be reduced, or whether its contents can be displaced. If so, the case must be either a **hernia,** a **bilocular** or **communicating hydrocele,** or a **burrowing abscess.** If an intestinal note is found over the swelling, or if it feels granular or lumpy like omentum, there is no doubt about it being a hernia. The same applies, if its reduction is accomplished in a sudden manner. But if it is only displaced gradually and the swelling corresponds accurately to the direction of the inguinal canal, we must think of one of the forms of hydrocele just mentioned. If the swelling is more laterally situated and can only be incompletely displaced, and if, in addition, it is painful on pressure, we should think of a burrowing abscess and search for confirmation in the vertebral column.

Finally, if the swelling is reducible, but feels neither like intestine nor omentum, but like a smooth roundish body, the case is one of an inguinal testicle or a prolapsed ovary.

If, however, the swelling is irreducible, but is soft or tensely elastic in consistence, we must think, according to its position, either of an enclosed hydrocele in the inguinal canal, or of a burrowing abscess which admits of no displacement. In this connection it is worth mentioning that there is also such a condition as a *hydrocele in the female.*

A solid tumour is more probably an *inguinal gland,* especially if it is subcutaneous, of the shape of a bean, and multiple. The examination of the appropriate lymphatic district will show whether its origin is due to cancer, hard or soft chancre, or merely a simple herpes genitalis.

Should no such origin be discoverable, there arises the question of *tubercle* or *lymphadenoma,* for the latter occasionally starts in the inguinal region. We have already discussed the differential diagnosis in the chapter on Tumours of the Neck.

If the swelling consists of a large uniform tumour which has grown rapidly and soon becomes immovable, it must be regarded as a **sarcoma.** If its size increases but slowly, it must be regarded as a **fibroma of the abdominal wall.** A movable, spindle-shaped, or cylindrical, hard tumour in the inguinal canal of a woman is most probably a **fibro-myoma of the round ligament.**

(4) LUMBAR REGION.

A swelling which appears in the lumbar region during an abdominal strain is a **lumbar hernia,** whether it is spontaneously reducible or only by pressure.

There are two sites for hernial protrusion in the lumbar region. One is at the outer border of the quadratus lumborum, just under the twelfth rib (Grynfeldt), the other is at Petit's triangle, the three sides of which are formed by the iliac crest, the external abdominal oblique, and the latissimus dorsi. Congenital lacunæ in the muscles seem, however, to be of more importance.

In certain rare cases of *infantile paralysis* the muscular atrophy may affect the muscles in front of the quadratus lumborum, which become paralyzed and atrophic over a definite limited extent. The border of the paralyzed area is so sharp that it imparts the same sensation as the boundaries of a genuine hernial ring. Fig. 208 depicts such a case; one of the first in which this paralysis was shown to be the cause of the pseudo-hernia.

A lumbar swelling, which is only partially reducible after steady pressure, is either a **burrowing abscess** or a **tubercular perinephritic**

abscess, which has burst through, behind. An examination of the urine will differentiate between these two possibilities. If the tumour is soft, but quite irreducible, with a perfectly smooth surface, and a more or less distinct fluctuation, it is a cold abscess with the origin just mentioned, or it may have started in one of the lower ribs. If it has a lobulated structure, and its situation is definitely subcutaneous, it must be regarded as a **lipoma.**

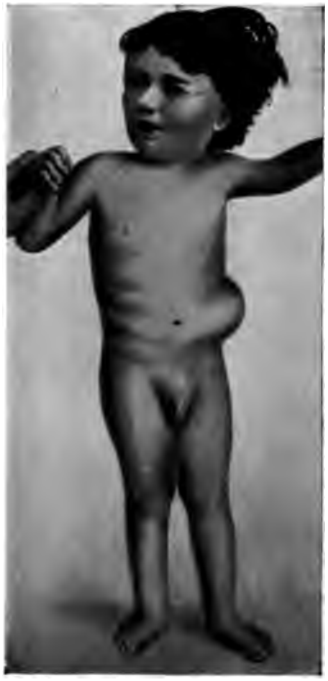


FIG. 208.—Pseudo-hernia in the lumbar region, due to localized muscular paralysis.

(5) SWELLINGS AND TUMOURS IN ATYPICAL POSITIONS.

If a swelling is not in one of the usual typical positions, but still possesses the features of a **hernia,** we are bound to assume a traumatic origin for it. Such trauma is almost always a laparotomy wound, which at once reveals itself by the scar. It is much more rare for such localized destruction of the abdominal wall to be due to accidental injuries or inflammatory processes.

The very rare abdominal hernia which appears at the outer border of the rectus muscle, in the vicinity of the linea semicircularis, should be distinguished from other abdominal hernia because of its rather typical occurrence.

Irreducible tumours, connected with the *skin* and *subcutaneous tissue,* are **lipomata,** more rarely soft **fibromata,** occasionally also **nævi which have become sarcomatous.** (See under "Tumours of the Back.") If they are more deeply situated and are connected with the *muscular layer of the abdominal wall,* they may be either fibromata of the abdominal integument, which have been previously mentioned, or *tubercle* of the muscle, which not infrequently attacks the abdominal *muscles.* The female sex, and a spindle-shaped, sharply-defined, uniformly hard tumour, are points in favour of a fibroma; but an irregular shape, partial softening, slight mobility and relaxation of the abdominal integuments are points in favour of tubercle (fig. 209).

If a hard fibrous tumour appear in the operation area months or even years, after an abdominal operation—e.g., the radical operation for hernia—we shall probably find a few threads of silk floating about in a little deeply situated pus, or embedded in granulations, as first shown by Schloffer.

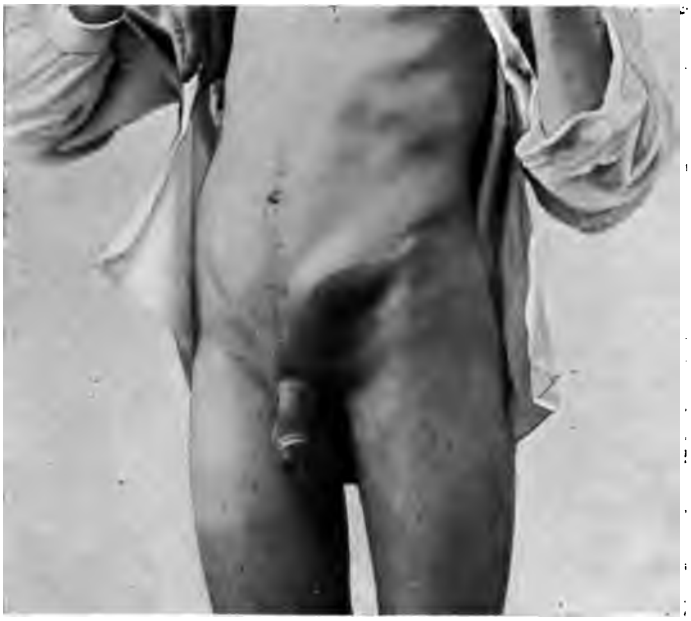


FIG. 209.—Tuberculosis of the abdominal wall.

Experience shows that a hard, board-like swelling in the ileo-cæcal region, which gradually reddens the skin and eventually produces sinuses, is most probably **actinomycosis,** originating in the cæcum. Every localized inflammatory swelling of the abdominal wall should be referred to infection breaking through from the intestine, but it is more likely to be tubercle or cancer than actinomycosis, except in the ileo-cæcal region.

CHAPTER L.

ABDOMINAL SINUSES.

A SINUS may arise anywhere in the abdominal wall, as the result of an abscess or a malignant growth breaking through. There is, however, nothing typical about such an occurrence. We will, therefore, confine ourselves here to sinuses whose position and characteristics are of diagnostic significance.

Most sinuses originate at the **umbilicus,** the point at which most abdominal organs meet in their embryological history, and which is the weakest spot in the abdominal wall, as far as later morbid processes are concerned.

(1) **Congenital umbilical sinuses** may be connected through a patent vitelline duct with the small intestine, or through a patent urachus with the bladder. The distinction is quite easy, because in the former cases fæcal matter escapes from the sinus, in the latter, urine. It is somewhat more difficult to account for a third umbilical sinus, which discharges neither fæces nor urine, but a thin mucoid fluid. This variety leads either into a piece of urachus which is open at the umbilicus, but is closed towards the bladder, or into a piece of vitelline duct, closed towards the intestine. As far as observations have gone hitherto, the latter is the more frequent termination.

These incomplete vitelline sinuses discharge a peculiar acid secretion like the gastric juice, and were formerly regarded as gastric fistulæ. The secretion sometimes causes digestion of the skin, and ulcerates the area surrounding the sinus.

(2) We diagnose an **acquired umbilical sinus** from the nature of the secretion, and thus differentiate pure suppurating fistulæ, biliary fistulæ, fæcal fistulæ, and urinary fistulæ.

(*a*) *Suppurating fistulæ* usually result from an intra-abdominal inflammatory process, which has burst through the abdominal wall at the umbilicus, i.e., its weakest spot. As a rule, it occurs in peritonitis which has become chronic (pneumococcus infection, fig. 177). A localized tubercular peritonitis may exceptionally burst through at the umbilicus.

Rupture of a suppurating hydatid or ovarian cyst may be mentioned as a very rare cause of suppurating fistula at the umbilicus. Empyema of the gall-bladder may occasionally open at the umbilicus, and cause a suppurating fistula, as long as the cystic duct remains closed. Finally, the sub-umbilical abscess at the back of the abdominal wall, previously referred to, may escape through the umbilicus.

A carefully passed probe will attain a certain depth in all these forms of umbilical sinus. But if, after several attempts, the probe does not reach beyond the umbilicus, the case is probably one of an *umbilical concretion* enclosed in a cutaneous pouch, or a ruptured *sebaceous cyst* or *dermoid* of the umbilicus, or, finally, a very small sub-umbilical abscess. If the microscopic examination of the secretion reveals chiefly detritus and epithelial cells, one of the first three possibilities should be thought of, but its accurate differentiation cannot be made out until the sinus is laid open. If the secretion is entirely purulent, we should think of sub-umbilical abscess.

(*b*) *Biliary fistulæ* arise in the manner previously indicated if the cystic duct regains its patency after rupture of the empyema of the gall-bladder.

(*c*) *Gastric and intestinal fistulæ*, which are easily recognizable by the character of their secretion, are caused by rupture of an ulcer. In the case of the stomach, this is either a simple gastric ulcer or cancer; in the case of the intestine it may be due to cancer or tubercle, but it may also be the consequence of a strangulated gangrenous umbilical hernia. The latter origin would at once be clear from the history.

(*d*) *Urinary fistulæ* may arise through extension of cystitis into a persistent urachus, with eventual rupture at the umbilicus. In other cases they may be due to the rupture of a phlegmon of the abdominal wall caused by infiltration of urine.

Typical sinuses are also found in the inguinal region arising from a strangulated hernia, or through rupture of a burrowing abscess. The character of the discharge from the sinus (intestinal contents or pus) and the previous history will lead to a correct conclusion. In addition, a sinus situated very much at the side points to a burrowing abscess. But if the sinus is in a more central position, either between both recti or at the outer border of one of them, there may arise a question of tubercle of the pubic bone, or of osteomyelitis with a sequestrum. Finally, urinary fistulæ after strictures may occasionally wander into the lower abdominal region.

We shall not discuss *ectopia vesicæ*, because this malformation cannot be mistaken for anything else.

CHAPTER LI.

EXTERNAL INGUINAL HERNIA.

ALTHOUGH abdominal herniæ are matters of daily occurrence, and the more common forms are correctly diagnosed by the public as well as by the profession, nevertheless there are some points worth referring to, even in this region. We begin with some observations on the subject of a "tendency to hernia," an expression which to many people conveys no clear conception.

A tendency to hernia implies certain anatomical conditions, which may lead to the development of hernia when the intra-abdominal pressure is raised, i.e., *even to the slightest temporary entrance of any of the abdominal viscera into a process of peritoneum.* A tendency to hernia may involve either the peritoneum or the muscular wall of the abdomen, or both.

In the former case the hernial sac is small, very narrow and congenital, being due to the imperfect obliteration of the *processus vaginalis* of the peritoneum. The sac is too narrow to admit any of the abdominal contents (fig. 210, *a*). The development of the muscles and aponeurosis may be quite normal.

In the latter case the primary change consists of a congenital or acquired weakness of muscles and fasciæ, combined with abnormal width of the canal. Every act of coughing presses the peritoneum, which is quite closed in the normal manner, against the unresisting internal inguinal ring, and causes it to bulge therein in a conical form (Kocher) (fig. 210, *b*).

Finally, both conditions, patent *processus vaginalis* and weak abdominal walls, may occur together.

The first variety of "tendency to hernia" cannot be demonstrated clinically as long as it is really only a "tendency." In the second variety of tendency the finger introduced into the inguinal canal meets with the well-known impulse. This also applies to the combined variety, in which the "tendency" rapidly develops into a complete hernia.

There is no doubt that the congenital tendency to hernia, in the form depicted in fig. 210 *a*, is of more importance than the acquired form, but this is no justification for throwing any doubt upon the occurrence of the latter.

After these preliminary remarks we now turn to simple inguinal hernia, dealing first with the form which manifests nothing abnormal externally.

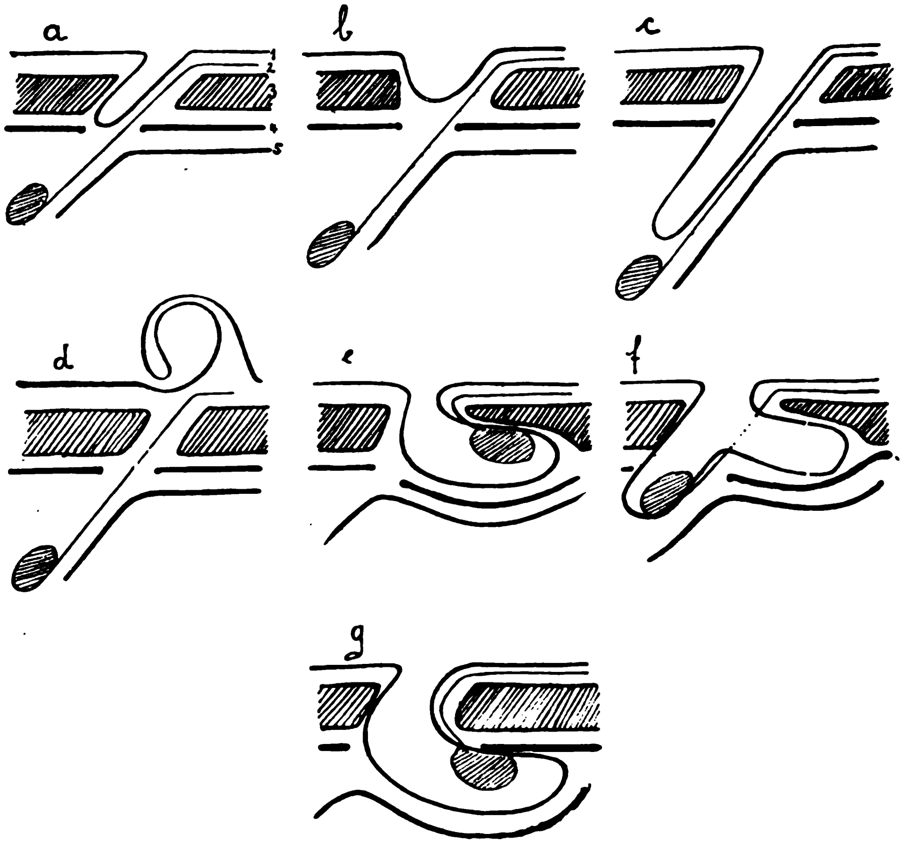


FIG. 210.—Relations between external inguinial hernia and the abdominal wall.

(1) Peritoneum. (2) Spermatic cord. (3) Muscular layer (especially internal oblique). (4) Aponeurosis of external oblique. (5) Skin.

(*a*) Congenital tendency to hernia.
(*b*) Acquired tendency to hernia with weak muscles.
(*c*) Fully developed inguinal scrotal hernia.
(*d*) Pro-peritoneal hernia.
(*e*) Intermuscular hernia (interstitial).
(*f*) Intermuscular bi-locular hernia.
(*g*) Subcutaneous hernia.

(1) DIAGNOSIS IN THE ABSENCE OF A HERNIAL SWELLING.

To examine a patient for hernia, when there is nothing visible externally, we place him in a standing posture, with his legs somewhat separated, and direct him to cough or press, while we see whether any bulging occurs. If the whole region above Poupart's ligament becomes pushed forward in a diffuse manner, without any visceral projection, we term the condition "soft groin," i.e., a congenital or acquired weakness of the abdominal wall. But if, on feeling both groins simultaneously, we appreciate a definitely circumscribed impulse on one side we may conclude that a hernia is beginning.

Then we invaginate the scrotal skin into the inguinal canal with the index finger, and the patient again coughs. In normal conditions this manœuvre causes the posterior superior limit of the inguinal canal to become more tense owing to contraction of the internal oblique ; but if there is any tendency to hernia a soft bulging forwards of the posterior wall of the canal will be felt.

If any of the intestinal contents have entered the canal it is no longer a matter of tendency to hernia, but of a hernia actually begun. It is advisable to let the patient lie down before making any further examination into the direction and range of the inguinal canal. If the abdominal contents remain within the canal after the abdominal pressure has been relaxed, but do not emerge from the external ring on subsequent coughing, the case is one of interstitial, or, better, of inter-muscular hernia.

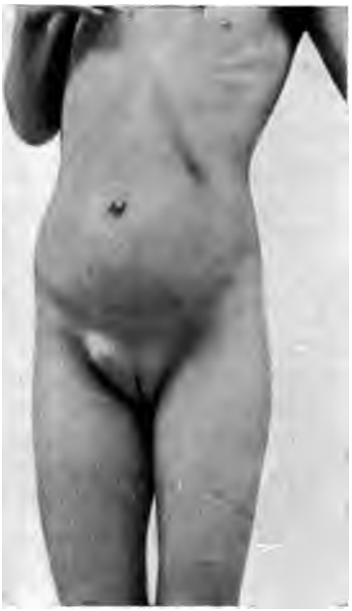


FIG. 211.—External inguinal hernia (at external abdominal ring).

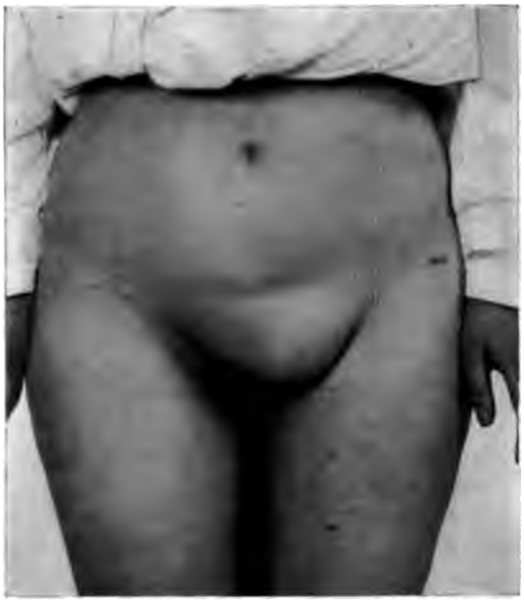


FIG. 212—External inguinal hernia, extending into the labium.

It sometimes happens that we are unable to secure any exit of intestine at the first examination, although a hernia is present. In such cases we may derive some assistance from carefully feeling the spermatic cord. If it is definitely thickened on one side, and if it is possible to demonstrate the presence of a narrow transverse pad, we may assume the existence of a hernial sac. This narrow pad represents the ring-shaped thickening which is often observed in older hernias, and which had been situated previously at the internal inguinal ring. If this examination is inconclusive, it is necessary to repeat the examination by making the patient lift a heavy weight, with his legs outspread and his body bent backwards.

It is not easy to demonstrate the presence of an inguinal hernia in *females* if it does not happen to be down at the time of examination. The narrowness of the inguinal canal does not allow of the introduction of the finger, as in the case of a male. If no impulse is seen or felt on coughing, we must endeavour to feel the hernial sac, which can be done more easily than in boys or men, because the round ligament in females disturbs us less than the spermatic cords in males. We place the index fingers on each side, median to the external inguinal ring over the pubic bone and move the skin over the latter, upwards and downwards, comparing the two sides, If a hernial sac is present, there is felt, not so much a thickening of tissue as fine friction caused by the gliding of one serous surface on the other. If this sign can be demonstrated at repeated examinations, we may be quite secure in our diagnosis of inguinal hernia.

This simple examination will sometimes explain severe attacks of abdominal pain which have been attributed to appendicitis, movable kidney, and other possibilities before the discovery of the hernia.

(2) DIAGNOSIS OF INGUINAL HERNIAL SWELLINGS.

If an abnormal swelling bulges forward in the inguinal region, the examination is a much simpler matter. If the swelling can be displaced backwards, and probably also yields an intestinal note on percussion, it is an interparietal intestinal hernia. If it is irreducible but yields a definite intestinal note, the diagnosis is the same. If the swelling, whether reducible or not, feels like a soft granular mass, it is an interparietal omental hernia. But if, on the other hand, we define clearly a smooth, roundish body, it must be an inguinal testicle in a male, an ovarian hernia in a female.

Every now and then, such a swelling is a testicle, although the patient has a plait and bears a girl's name. A *pseudo-hermaphrodite* is proclaimed by a somewhat enlarged clitoris (fig. 213), but possessing both testicles and a vagina, the patient is too much of a male to be a woman, and too much of a female to be a man. It is fortunate if this state of affairs is discovered before marriage is contracted. Some of these pseudo-hermaphrodites celebrate the discovery of their sex by indulgence in tobacco and alcohol ; others, despite their testicles, retain the sensitiveness of the female and remain true to their frocks.

The detection of an inguinal testicle does not, of course, exclude a hernia; indeed it renders the presence of a hernia very probable.

These herniæ may be divided into three main forms, according to their position :—

(1) *Pro-peritoneal hernia*, situated immediately under the parietal serous membrane (fig. 210, *d*).

(2). *Inter-muscular hernia*, in the area of the muscular abdominal wall, generally between the internal oblique muscle and the aponeurosis of the external oblique. (Interstitial hernia in a narrow sense; subaponeurotic hernia.) (Fig. 210, *e* and *f*, and fig. 218).

(3) *Subcutaneous hernia*, between the aponeurosis of the external oblique and the skin (inguino-superficial hernia, sub-fascial hernia). (Fig. 210, *g*, and fig. 218.)

All these herniæ may occur in the form of herniæ with divided sacs, which unite into one hernial sac in the scrotum or labium. They all occur, but much more rarely, in the female sex, both in the simple form or with divided sacs (fig. 214).

In males they are often associated with a retained inguinal testicle. If the hernial sac is of the

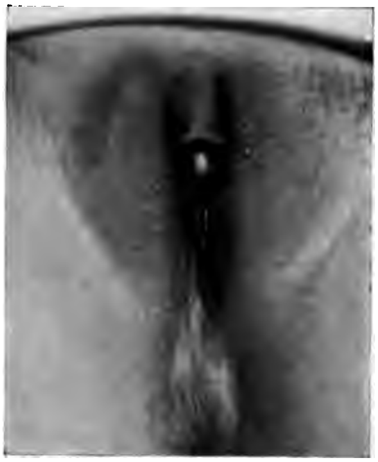


Fig. 213—Genitalia of a male pseudo-hermaphrodite, with a vagina, and with testicles in a hernial sac.

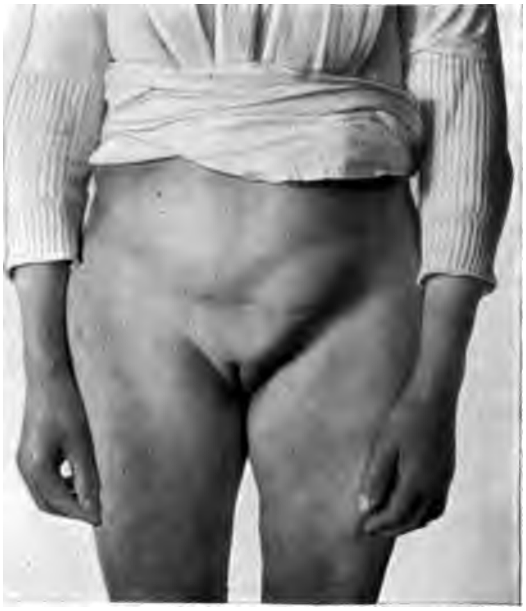


Fig. 214.—External inguinal hernia with a divided sac (one portion being labial, the other intermuscular, filling and emptying separately).

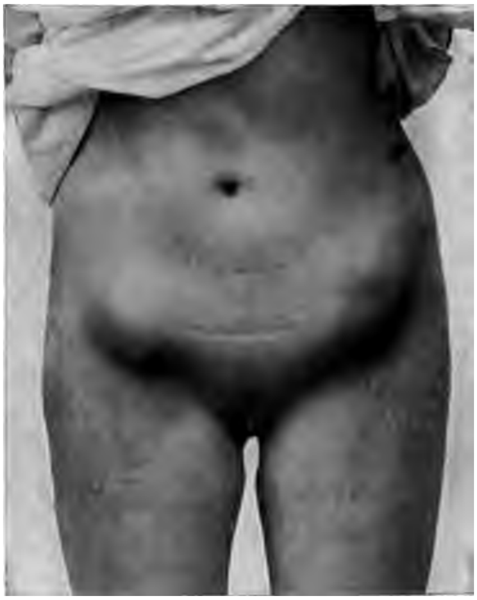


Fig. 215.—Bilateral burrowing abscess due to spinal caries.

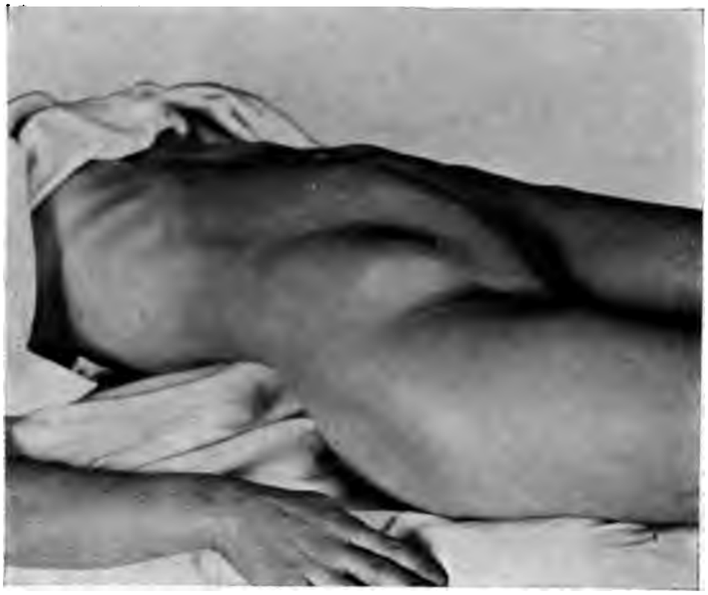


Fig. 216.—Right-sided burrowing abscess, admitted into hospital as " hernia."

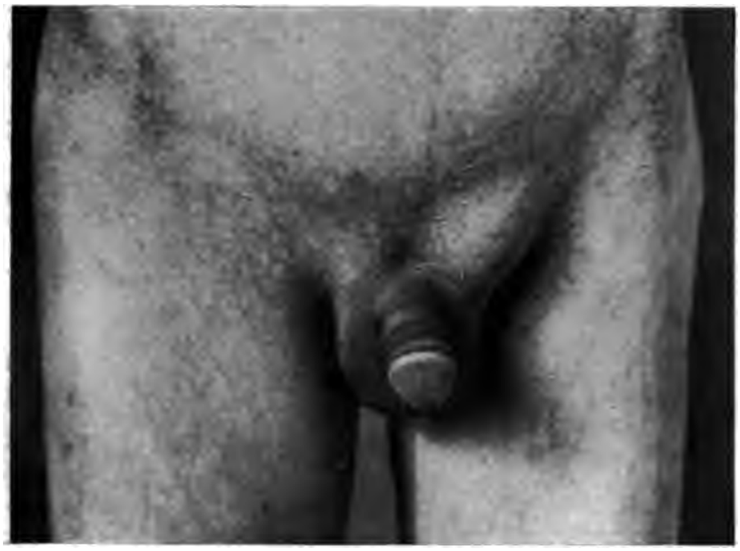


Fig. 217.—Hydrocele of spermatic cord.

subdivided variety the testicle is often found lying in the upper interparietal portion.

As far as the clinical diagnosis of these various forms is concerned, the *pro-peritoneal* variety is, as a rule, detected first when it becomes strangulated. The symptoms are those of internal strangulation, and a roundish resistance can be felt deep down behind the internal inguinal ring. The diagnosis of the *intermuscular* and the much rarer *subcutaneous* varieties, has already been referred to in common; but their differentiation is of some interest. For this purpose, the patient is directed to sit up without supporting himself by his arms. If the aponeurosis of the external oblique then becomes tense *over* the hernia, the latter is intermuscular (fig. 218); in other cases

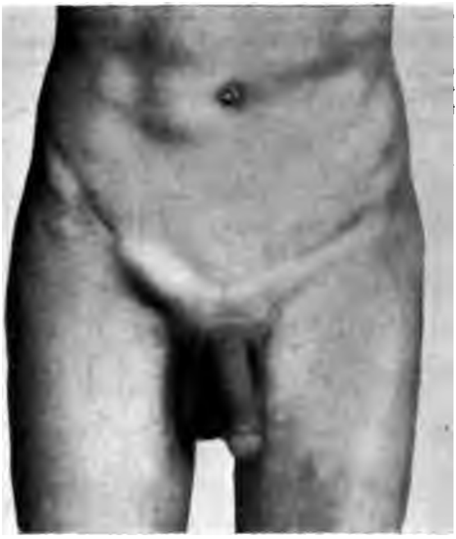


FIG 218.—Intermuscular inguinal hernia, under the aponeurosis of the external oblique.

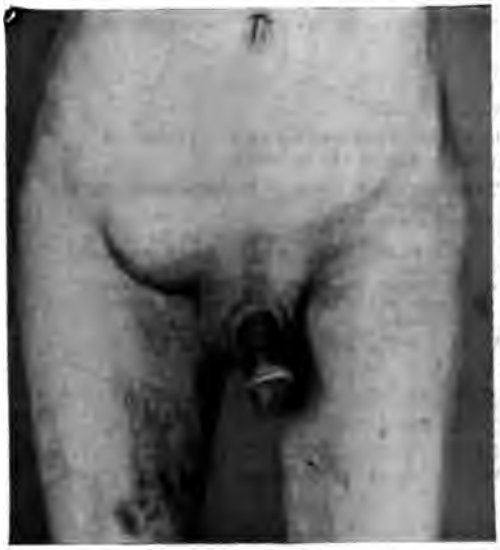


FIG. 219.—Subcutaneous inguinal hernia (H. inguino superficialis) situated under the skin.

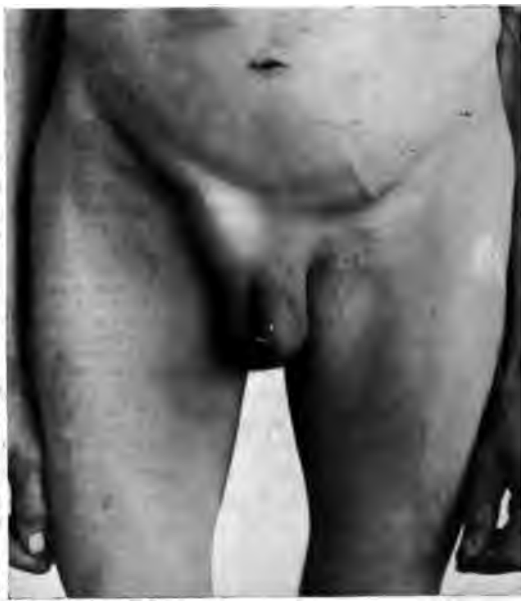


FIG. 220.—External inguinal hernia, just come through the external ring.

it is subcutaneous (fig. 219). In this illustration the laxity of the hernia indicates its subcutaneous position at the first glance.

The following case is typical of a hernia with a divided sac :—

I operated on a man, aged 68, for what was apparently an ordinary scrotal hernia, without opening the inguinal canal, but I excised the sac as high up as possible. All went well until the patient stated, three weeks subsequently, that the hernia had returned. As a matter of fact, protrusion of the intestine could be seen above the internal inguinal ring, when the patient coughed. A second operation revealed an intermuscular hernial sac running laterally under the aponeurosis of the external oblique. It was quite as large as its scrotal offshoot, which had been removed three weeks previously, but was overlooked at the operation, because it had not been felt at the previous examination.

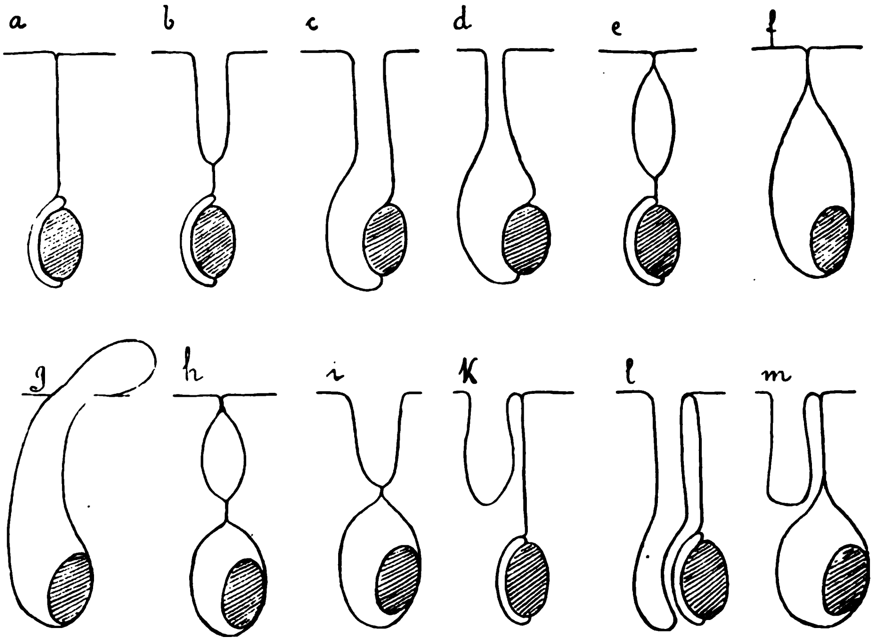


FIG. 221.—Diagrammatic representation of the relations between the vaginal process of peritoneum, in congenital and acquired herniæ and in hydrocele.

(*a*) Normal obliteration of the process
(*b*) Partial patency of the process. Funicular hernia.
(*c*) Complete patency of the process. Testicular hernia.
(*d*) ,, ,, ,, ,, with narrow neck, communicating hydrocele.
(*e*) Patency in centre of process. Funicular hydrocele.
(*f*) Patency at lower part. Testicular hydrocele.
(*g*) Outgrowth of *f* upwards. Bilocular hydrocele.
(*h*) Combination of *e* and *f*. Funicular and testicular hydrocele.
(*i*) Combination of *b* and *f*. Funicular hernia and testicular hydrocele.
(*k*) Acquired funicular hernia.
(*l*) ,, ,, ,, reaching as far as the testicle, and imitating (*c*) congenital testicular hernia.
(*m*) Combinat on of *f* and *k*. Acquired funicular hernia, and congenital or acquired testicular hydrocele.

Simple as the diagnosis of interparietal hernia would appear to be from the foregoing remarks, nevertheless mistakes do occur. Femoral herniæ, with processes extending over Poupart's ligament, are especially liable to be mistaken for inguinal herniæ and *vice versa* (figs. 231 and 232). But it is not only with other herniæ that confusion arises, but also with other diseases. Thus trusses have been ordered for burrowing abscesses, and not by quacks only, for medical practitioners sometimes neglect to make an examination from misplaced motives of delicacy, or conduct it so indifferently that it is quite useless.

Such errors are easily avoided by careful examination. As a rule, a spinal abscess shows fluctuation ; a hernia does not. It is true that the pus can sometimes be displaced, but this only occurs gradually after steady pressure, and not suddenly with a gurgle, as in the case of the intestine. When the pressure is relaxed the swelling gradually returns without any straining, whereas a hernia requires some change of posture or the straining of abdominal pressure before it makes its re-appearance. Finally, most burrowing abscesses are situated more laterally than inguinal herniæ usually are (figs. 215 and 216).

(3) DIAGNOSIS OF LABIAL AND SCROTAL HERNIA.

A swelling situated within the scrotum or labium can only be a hernia if it has a pedicle running in the inguinal canal. If this be not the case (fig. 222), there is no hernia. If, however, there be such a pedicle, we must see whether the swelling is reducible (fig. 223). If the swelling can be displaced backwards, possibly with a buzzing or gurgling sound, the case is one of hernia. If the reduction is slow and requires steady pressure, the condition is usually one of communicating hydrocele, and only rarely a bilocular hydrocele with the second sac in the abdomen.

A varicocele is apparently reducible, but the filling up of the swelling like a handful of earthworms, when the patient stands, and its immediate relaxation when he lies down, without, however, any of its contents suddenly receding, ought to exclude the possibility of any confusion. If the swelling has a pedicle, but is irreducible, the case may be one of testicular hydrocele reaching as far as the inguinal canal. Such a swelling is, however, uniformly tense and elastic, is dull on percussion, and generally translucent, whereas an irreducible, unstrangulated intestinal hernia is not translucent, is of more lax consistence, and usually gives the intestinal note on percussion. A strangulated hernia is indeed also tense like a hydrocele, but it has a smaller and very tender pedicle, and, moreover, is always attended by symptoms of intestinal obstruction. But the following case will indicate how error can arise.

A young man sought advice for a classical pear-shaped irreducible testicular hydrocele, with a narrow neck in the inguinal canal. He stated that he had felt, two months ago, just before the appearance of the hydrocele, a sudden pain in the left hypogastrium. We attached no importance to this, and adhered to the diagnosis. At the operation, a hydrocele was indeed discovered, but it had a narrow offshoot in the inguinal canal, containing a small adherent plug of omentum. The patient, therefore, had a small strangulated omental hernia in a congenital sac, and the fluid was really hernial in character. The pain in the hypochondrium was due to the dragging of the omentum at the moment of strangulation. It is important to note this type of case, because its occurrence is not infrequent.

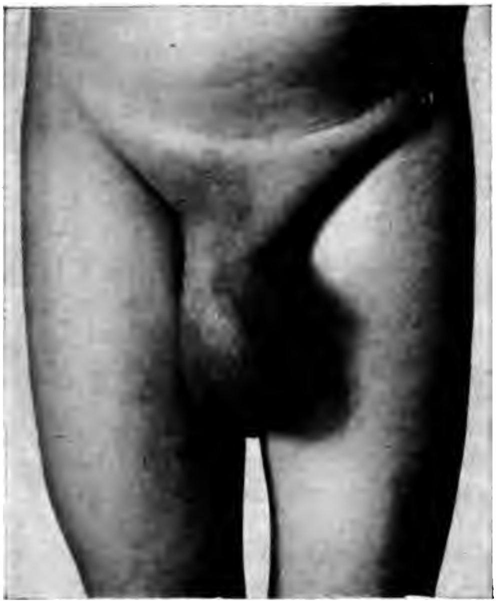


FIG. 222.—Testicular hydrocele.

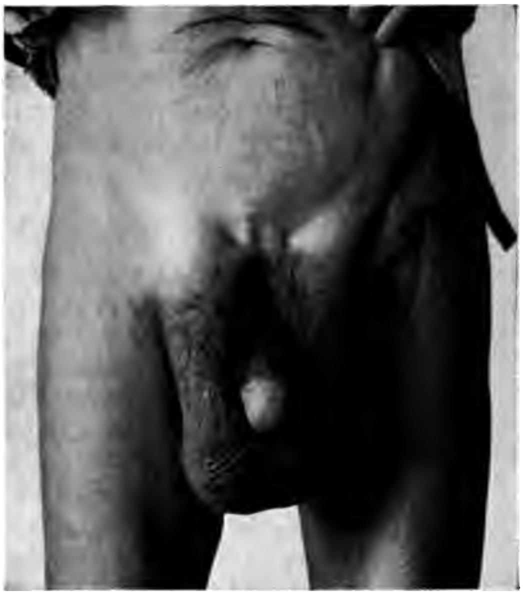


FIG. 223.—Bilateral external scrotal inguinal hernia.

An irreducible granular or lobulated scrotal swelling which possesses a pedicle may be one of three things: (1) Omental hernia; (2) hernial sac, with much peri-hernial fat; or (3) a lipoma of the spermatic cord. Unaltered omentum in a hernial sac feels more finely granular than a lipoma of the spermatic cord. But this distinction is a very delicate one, and can only be appreciated by very experienced fingers. The history is really of more value in this connection. If the patient states that the size of the swelling is very variable, we should think of hernia, as also if he frequently complains of pain during digestion. The not infrequent cases wherein a small lipoma of the spermatic cord is present with a hernia are hardly capable of being diagnosed.

It is of diagnostic importance to know that inguinal hernia and lipoma of the spermatic cord are sometimes accompanied by obstinate spermatic cord neuralgia, the cause of which remains obscure until the hernia or the lipoma appears.

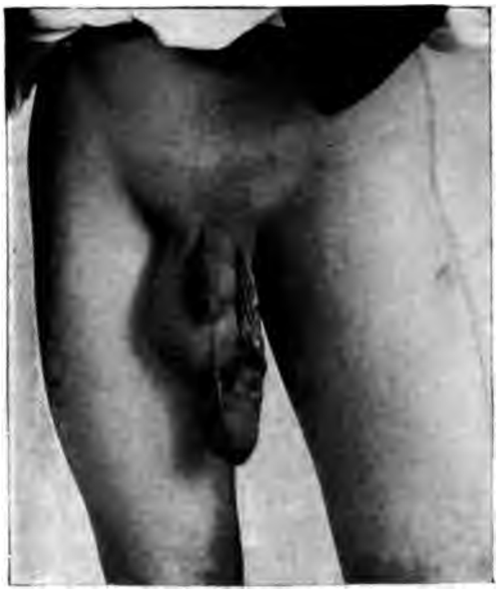


FIG. 224.—Varicocele with atrophy of testicle.

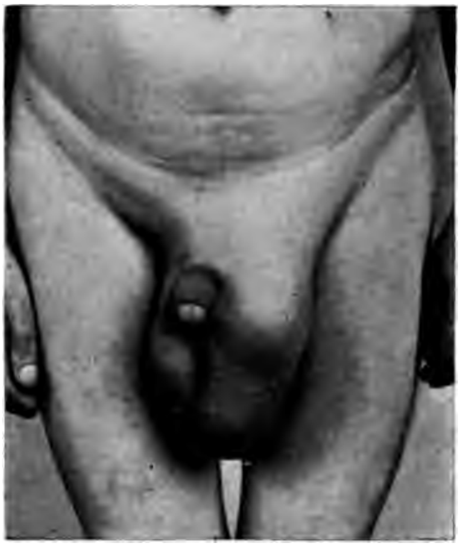


FIG. 225.—Left-sided hydrocele and external inguinal hernia.

Diffuse extension of peri-hernial fat is rarely found in external inguinal herniæ, but it is quite general in internal inguinal herniæ and in femoral herniæ.

CHAPTER LII.

INTERNAL OR DIRECT INGUINAL HERNIA.

THERE is usually no difficulty in distinguishing an internal direct inguinal hernia from an external indirect hernia, if one bears in mind that in the former the hernial ring leads directly into the abdominal cavity, and does not run through the inguinal canal. This form is frequently bilateral, and does not descend into the testicle, but remains above the root of the penis after giving rise to a pronounced transverse fold in this region (fig. 227). In contrast to the external variety, internal hernia usually occurs in the middle-aged and the old, and almost always in males. On coughing a semi-globular swelling appears, which is more sharply defined laterally than in the case of an external inguinal hernia.

No importance should be attached to the clinical demonstration of the position of the deep epigastric artery. It runs upward, along the inner side of an external hernia, and along the outer side of an internal hernia. Valuable as this sign may be at the operation, when a case is doubtful, it is, as a rule, impossible to palpate the artery before operation.

In addition to the cases which fit in the above scheme, there are two varieties of inguinal hernia which may present some difficulties in diagnosis:—

(1) External inguinal herniæ of old standing, wherein the canal has lost its obliquity, and the hernial ring opens directly into the abdominal cavity just as in the case of an internal hernia, but the hernial sac has not descended into the scrotum. The gradual protrusion of the swelling towards the side may, in these circumstances, be the only sign pointing to an external hernia.

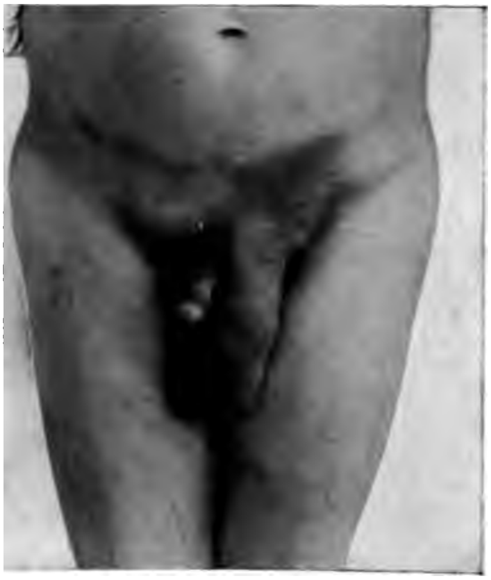


FIG. 226.—Internal inguinal hernia on the right, and external hernia on the left.

(2) Internal inguinal herniæ which descend to a small extent into the scrotum—a condition noted by Berger, and one which we have occasionally observed—and which are easily mistaken for external herniæ. Difficulty in diagnosis will be encountered if it is not possible, on examining the hernia after reduction, in a recumbent posture, with one finger in the canal and the other introduced directly into the ring, to demonstrate that there is still a bridge of tissue between the two fingers.

The question which arises occasionally in connection with a direct hernia, as to whether it is more of a lipoma or more of a hernia, is unimportant, because it is quite a relative matter. All internal inguinal herniæ have a more or less extensive fatty layer within the sac, and the personal equation enters in deciding where a hernia ceases and a lipoma begins.

A final word concerning **hernia of the bladder.** This may occur either in an external or an internal inguinal hernia, but more frequently in the latter. It will be understood that one cannot speak of hernia of the bladder, when a piece of this organ is dragged up into the ring during the course of a radical operation on the hernia. A portion of the bladder must be part of the regular contents of the hernia—not necessarily of the hernial sac, because the bladder often extrudes itself, extraperitoneally, along the side of the sac, to the ring. Disturbance of micturition would justify the suspicion of a hernia of the bladder, whether it be difficulty in emptying the organ, or frequent tenesmus. The suspicion would be confirmed it it were observed that these disturbances coincide with fulness of the hernia. It is possible to demonstrate in such cases that when the bladder is very full there is fluctuation in the hernia

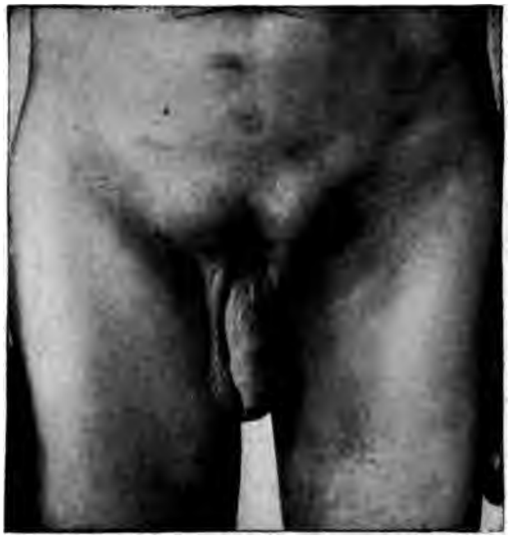


FIG. 227.—Bilateral internal inguinal hernia.

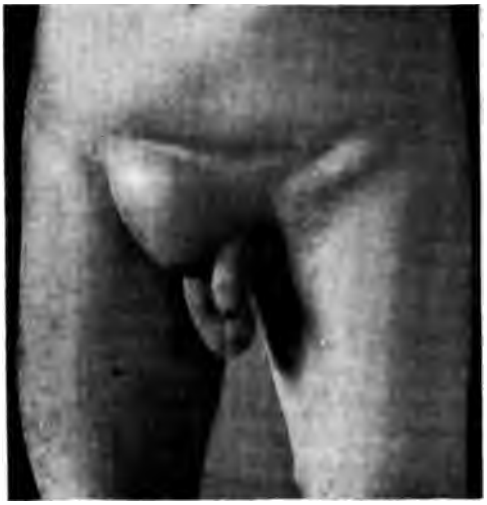


FIG. 228.—Internal inguinal hernia.

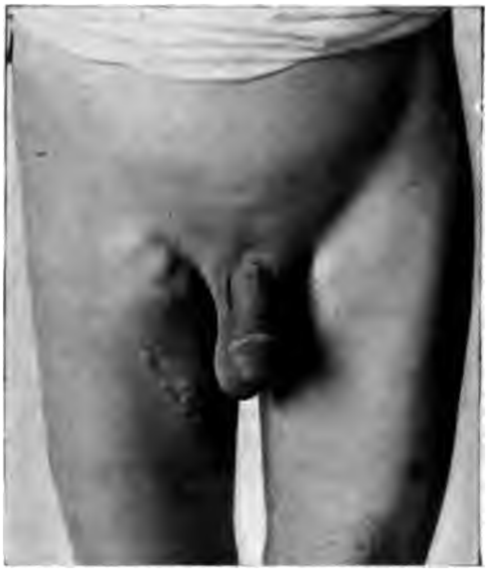


FIG. 229.—Bulging of the femoral region through varicose swelling of internal saphenous vein

and dulness over it on percussion, signs which disappear after the bladder is emptied. In some circumstances the catheter is required to empty the bladder, because internal inguinal herniæ happen to be most frequent in patients with enlarged prostates.

CHAPTER LIII.

FEMORAL HERNIA.

THERE are not many swellings which can be mistaken for femoral hernia. Errors may, however, possibly arise in connection with hernial protrusion of the saphenous vein, enlarged glands, lipomata, burrowing abscesses in the crural ring, and, finally, inguinal herniæ. We will consider these in order. The not infrequent spindle-shaped or saccular **protrusions of the saphenous vein,** if visible, glimmer with a blue colour through the skin, the slightest pressure suffices to make them vanish, but they return as soon as the pressure is relaxed. They react with mathematical, or rather with physiological, precision to every variation in venous pressure, such as is caused by coughing, vomiting, &c., and even to the normal breathing, in a recumbent position. These signs are so clear that confusion would apparently seem impossible, but as a matter of fact errors have arisen. **Burrowing abscesses** do not, as a rule, break through the spaces transmitting the vessels, but more laterally through a muscular interspace; they are frequently multilocular, and are displaced on gradual pressure, yielding a sensation of elastic resistance. They fill up again as soon as the pressure is relaxed, without any abdominal straining or change in the posture of the patient. But even in the cases wherein these abscesses appear towards the middle line, as in fig. 231, the other

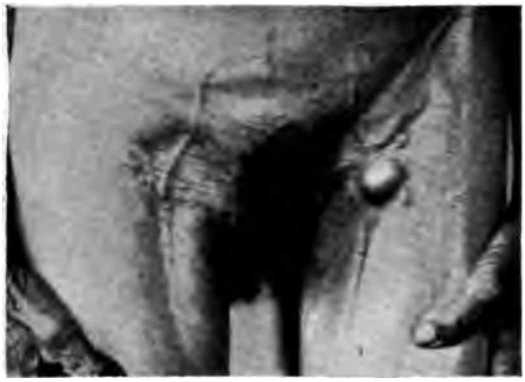


FIG. 230.—Varix of internal saphenous vein.

signs are so definite, than an error is hardly possible after careful examination.

Enlarged glands may present more difficulties. The entrance of the infection is usually found somewhere on the leg or foot. The chief characteristic of enlarged glands, even when chronic, is their sharp limitation at the femoral ring, whereas a hernia always possesses a pedicle which runs under Poupart's ligament, and which is compressible against the pubic bone. The rare subserous **lipomata** are also, strictly speaking, pedunculated, but the pedicle cannot be felt like the neck of a hernia. The absence of a palpable process into the abdomen, combined with the absence of any variation in volume and symptoms of hernial protrusion, would suggest a lipoma, behind which there may be a small pouch of peritoneum, although it may never have contained any intestine or omentum. It is very significant of femoral herniæ that a very small hernial sac is often surrounded by a large amount of fatty tissue, which grows into a lipomatoid structure.

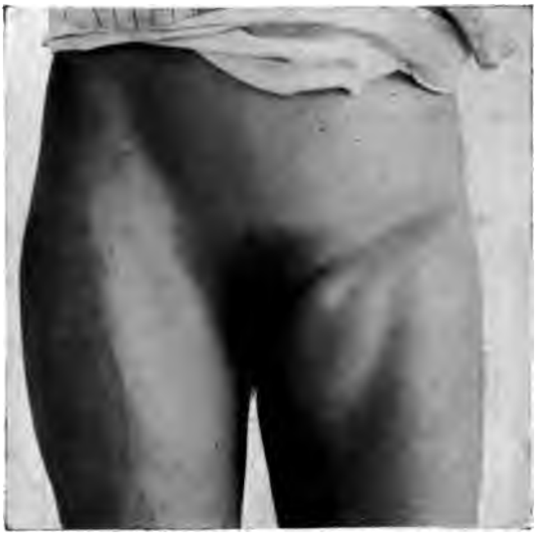


FIG. 231.—Crural burrowing abscess from spinal caries.

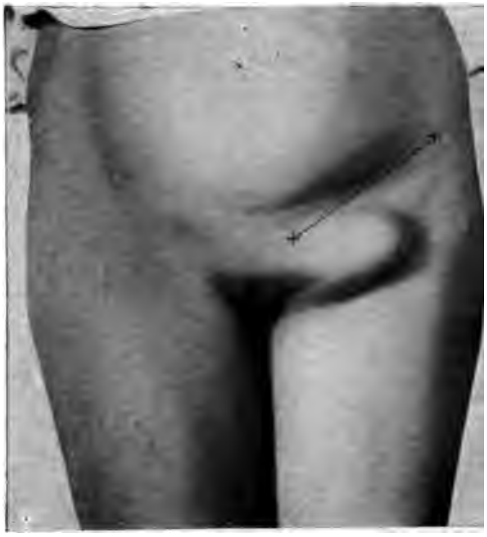


FIG. 232.—Crural hernia in a female. Its relatio Poupart's ligament, x—x.

Superficial lipomata also occur in this region, either as isolated growths, or as a partial manifestation of a general lipomatosis (fig. 234).

If the swelling has a definite pedicle which becomes lost under Poupart's ligament, the diagnosis of **hernia** can hardly be mistaken. But if there is no intestinal note, the case is either one of peri-hernial lipoma, just mentioned, or an omental hernia.

The differentiation depends less upon the physical condition found on palpation, than upon the presence or absence of symptoms of omental dragging, as already indicated in the case of inguinal herniæ. The question is, however, a matter of indifference, because if treatment is called for, operation must be undertaken in either case.

If a penndunculated swelling feels definitely elastic, there must be a collection of fluid in an old obliterated hernial sac. If attacks of sudden abdominal pain, without intestinal obstruction, have preceded the onset of a tense hernial swelling, it is obvious that a small piece of peritoneum has been strangulated in the ring and has led to the development of hernial fluid.

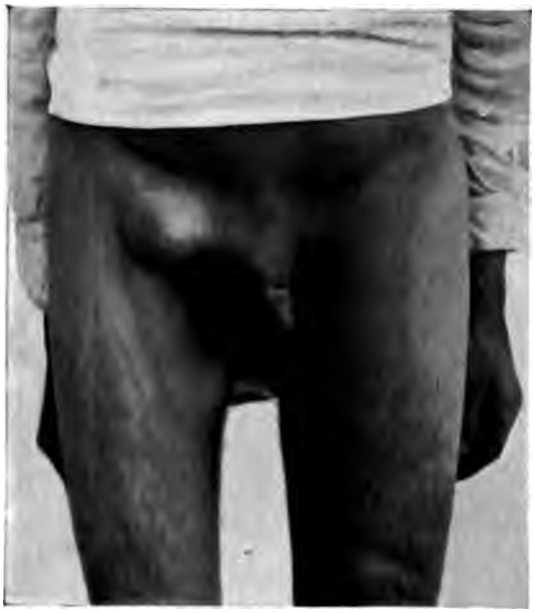


FIG. 233.—Femoral hernia in a male.

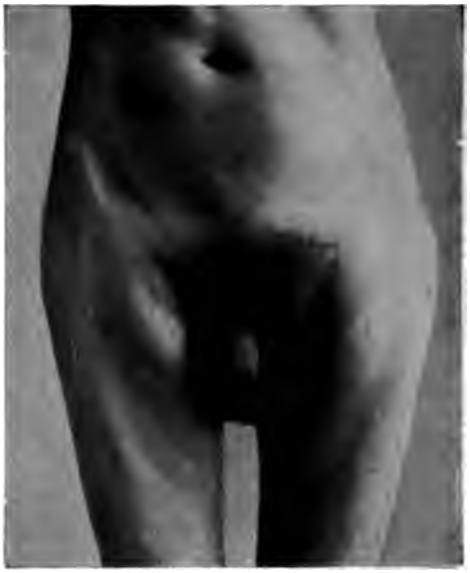


FIG. 234.—Symmetrical lipomata in crural region.

Having determined that the swelling is a hernia, the question still remains as to whether it is really a **femoral hernia.** If the tumour is clearly below Poupart's ligament, the matter is settled ; but if it is riding on the ligament the solution is not so clear. It may be an inguinal hernia which has wandered downward, or it may be a femoral hernia which has grown upwards (figs. 236 and 237). The differentiation is not difficult, if we are able to reduce the hernia. We base our decision on the position in which it recedes, and by feeling the ring. It is, however, otherwise, if the hernia is irreducible, whether it be strangulated or not. Incorrect diagnoses are very frequent in such cases, and as a rule a femoral hernia is taken to be an inguinal hernia.

As Poupart's ligament cannot be clearly defined because it is overlain by the hernia, and because it is obscured by fat in elderly women, Malgaigne's line has been adopted to indicate its position, i.e., the line joining the spine of the pubis with the anterior superior spine of the ilium (fig. 232). Everything above this line is to be ascribed to inguinal hernia, and below it, to femoral hernia. But this criterion is not always reliable. More importance is to be attached to the position and direction of the neck of the hernia, which can generally be felt on careful palpation, and which is recognizable, when strangulated, by its size and tenderness on pressure. If it runs vertically and is capable of being moved from side to side on the crest of the pubes, or if it appears to run deeply down when the hernia is displaced upwards, the case is one of femoral hernia. If it runs outwards and upwards, or directly outwards, the case is one of inguinal hernia. This sign enables a definite diagnosis to be made in such a case as fig. 237, although the major portion of the hernial swelling was above Poupart's ligament.

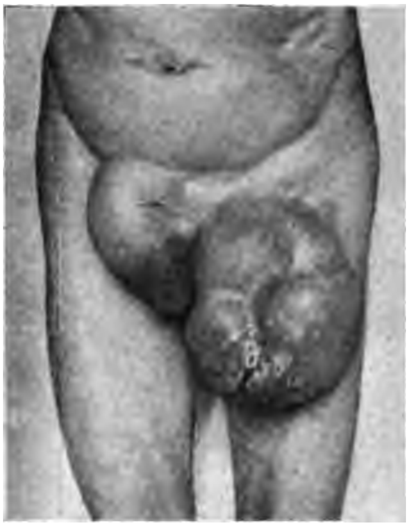


FIG. 235.—Large femoral hernia on the left. Direct inguinal hernia on the right.

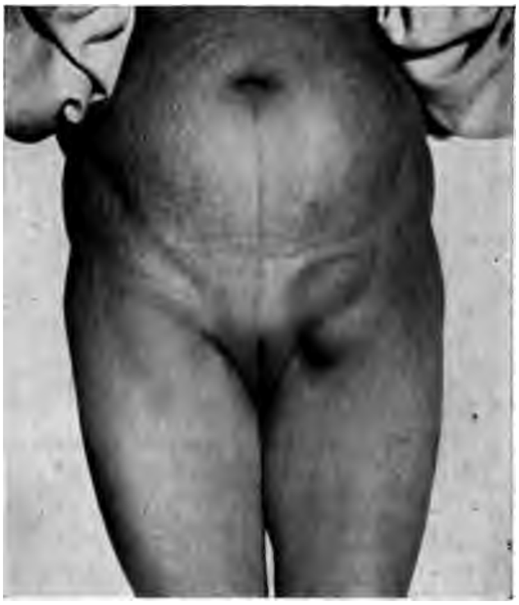


FIG. 236.—Bilocular femoral hernia encroaching above Poupart's ligament.

If, despite a history of hernia, we find nothing at our first examination, we must carefully compare the two sides for slight tissue thickening, or for fine friction, as already mentioned in connection with inguinal hernia. We must especially observe whether the area of the fossa ovalis is fuller on one side than on the other. In this way we may sometimes discover the explanation for inexplicable abdominal pains, even if the patient denies any hernial protrusion. I have already mentioned that I once removed an appendix because of the history and the diagnosis of the family practitioner, and then, later on, recognized that the pains which continued were really due to a small concealed femoral hernia on the right side.

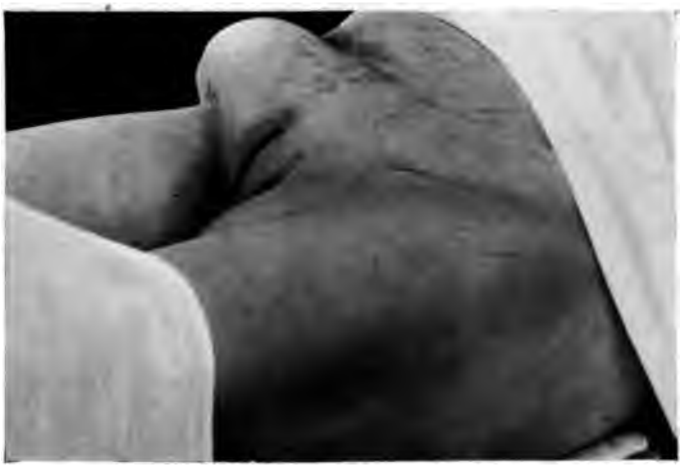


FIG. 237.—Strangulated femoral hernia projecting above Poupart's ligament.

There are a few *abnormalities* in connection with femoral hernia, which are generally only discovered at the operation, but which might be recognized, or at least suspected, on careful examination.

For instance, the hernial sac may pierce into the muscle under the *fascia of the pectineus*, and thus resemble an obturator hernia—a rare condition only occurring in women and designated after Cloquet. The hernia may descend *under the large vessels*, or it may appear to their *outer side* in the opening through which they run, or it may leave the abdominal cavity through a muscular interspace. This latter form, the so-called *Hesselbach's hernia*, has been recognized before operation by means of its broad base and its lateral position. Finally, we find here, as in inguinal hernia, a *pro-peritoneal* form, which does not come through the abdominal wall.

CHAPTER LIV.

TRAUMATIC HERNIÆ.

THE surgeon will occasionally be confronted with the question as to whether the hernia which he has demonstrated has arisen through an "**accident.**" The term is usually meant to include any sudden strain, which does not come within the patient's ordinary occupation, or one within his occupation, if it has overtaxed his strength. Most of these cases are accounted for by the sudden strain of abdominal pressure, with the body in disadvantageous posture.

The story of the Zouave trumpeter is typical of this. As he was blowing for the attack at Malakoff, he fell into a hole, and got up with a hernia.

We have to take into consideration both direct and indirect trauma. Neither form can cause a hernia in persons hitherto perfectly normal. But indirect trauma—rarely direct—may convert a tendency to hernia into actual hernia, i.e., the stretching of the pre-existing small hernial sac and the consequent dragging of the parietal peritoneum may determine the entrance of some intestine or omentum into the sac. This process is, as a rule, so painful, that it prevents any further bodily exertion, and the patient feels bound to seek medical advice. These two conditions are usually associated in the evidence of a "traumatic hernia," though there are exceptions varying with the occupation and energy of the workman.

A hernia which has arisen in this manner is small—at most the size of a hen's egg—the sac is thin, and not palpable; the hernial ring is generally small but it may be of medium size. The intestine does not always protrude when the patient assumes the erect posture, but once having protruded it does not always return when he lies down. The hernia is only slightly movable, without, as has been asserted, necessarily being strangulated. There should be no traces of a truss on the skin.

The consideration of these circumstances enable us to decide whether the hernia could possibly be traumatic, i.e., whether it could have arisen from sudden strain. As a rule, nothing more definite can be said, unless the patient happens to have been examined shortly before, specially for hernia. However reasonable the claim of the workman for compensation may be, for the genuine traumatic change of his "tendency to hernia" into an actual hernia—for instance, a radical cure by operation, without any money payment—nevertheless the practitioner should be extremely cautious in countenancing the fashion of making a profit out of these too easily assumed cases of traumatic hernia.

The extent to which these attempts may go is illustrated by the fact that even gonorrhœal epididymitis and inguinal buboes after soft chancres have been represented as "traumatic hernia."

Most so-called traumatic herniæ are of the external variety; but all forms have been at times ascribed to injury. We may, however, discard the possibility, in internal inguinal herniæ and umbilical herniæ, and should be very sceptical with femoral herniæ. An epigastric hernia is much more likely to be due, in some considerable degree, to trauma.

CHAPTER LV.

STRANGULATED HERNIA.

It is most important to observe that *strangulation* must not be confused with *irreducibility*, for it is only one special variety of irreducible herniæ. Both intestine and omentum may be *irreducible* without being strangulated; the cause usually is the presence of adhesions. Moreover, the omentum within the sac may develop so much, entirely of its own accord, that it is unable any longer to slip back through the hernial ring, even if no adhesions exist.

A special form of irreducibility is found in connection with certain herniæ of the cæcum and ascending colon, and also of the sigmoid and descending colon. In these cases, not only does the intestine, which is covered by peritoneum, leave the abdominal cavity, but also its extraperitoneal site of attachment, and, in consequence of the slipping down of this surface of attachment into the area of the hernia, the intestine loses all its tendency to return within the abdomen. Thus it comes about that there are herniæ of the large intestine with very little hernial sac—a point to be borne in mind at the operation.

There are also those enormous herniæ which are met with less frequently than formerly, which, apart from adhesions, are irreducible, because the abdomen no longer contains enough space to receive them. Petit aptly said that these herniæ "have lost their right of domicile."

The subjective symptoms of a non-strangulated but irreducible hernia are not, as a rule, any more severe than those of a free hernia. But dragging pains are often present; though they manifest themselves in the upper part of the abdomen rather than in the hernial region. Local pain usually arises if the patient maltreats the hernia with a truss.

Strangulation is a form of irreducibility which arises suddenly, and is attended by constriction of the pedicle, fixity of the presenting intestine, with disturbance of the circulation in the contents of the sac. A number of questions require answering in this connection.

(1) Is the Case really one of Hernia?

This is no superfluous question. Every surgeon has had infants referred to him with the diagnosis of strangulated hernia, when the case has been one of acutely arising *hydrocele in an infant.* The distinction is really not difficult. The tense hydrocele swelling, is as a rule, definitely limited above, and does not run into the inguinal canal. The infant passes motions and flatus and does not vomit, at any rate persistently. It takes the breast or bottle, again, after a short interval, an indulgence which an infant with a strangulated hernia never enjoys. It certainly cries, because the rapidly increasing effusion is a source of discomfort, but it does not appear to suffer severely. If one has made certain of the diagnosis, by careful examination, the swelling may be tapped quite safely and the anxious parents be convinced of the accuracy of the diagnosis.

Besides the hydrocele of young children, there are complications connected with an *inguinal testicle* which may resemble strangulated hernia, namely, *twisting of the pedicle* of the testicle and its *strangulation.* This error is all the more explicable because most inguinal testicles are associated with herniæ, and the patient will therefore give a history of hernia. Formerly, inflammation and strangulation of the inguinal testicle were the only recognized morbid conditions thereof, but since the time of Nicoladoni it has been agreed that torsion of the abnormally pediculated testicle is the usual affection, and the possibility of strangulation has been somewhat neglected. But that it exists appears from the following case :—

A patient who had a left-sided hernia, which, however, had not come out of the inguinal canal since his fourteenth year, felt a severe pain in the left inguinal region, on lifting a heavy weight. A tender swelling, which looked like a strangulated hernia at first sight, appeared. But the emptiness of the left half of the scrotum and the patency of the intestine enabled a correct diagnosis to be made, and the operation showed that the inguinal testicle had slipped down to the narrow external inguinal ring and had been conducted externally under the skin. The kinking of the spermatic vessels at the external abdominal ring, and their dragging owing to the abnormal position of the testicle, had led to the formation of a considerable infarct. In this case the diagnosis could only lie between torsion and strangulation with subcutaneous displacement. The subcutaneous position of the testicle contra-indicated simple torsion. If a testicle within the inguinal canal becomes twisted, it remains within the canal.

The symptoms of the more frequent condition of *torsion* are as follows: sudden severe pain and the appearance of a tumour. In addition, there are often reflex symptoms which suggest strangulated hernia, viz., severe abdominal pains, temporary obstruction to the passage of motions and flatus, and even collapse. In the inguinal region, there will be found a tender swelling, like a strangulated hernia. The empty scrotum and the return of the passage of motions and flatus after the cessation of the original reflex symptoms, usually enable a diagnosis to be made. But as it is impossible, at the beginning, to distinguish, with absolute certainty, between a twisted testicle in the inguinal canal and a strangulated interparietal hernia, and as surgical intervention is urgent in either case, it is bad practice to wait until the diagnosis is established by signs of intestinal gangrene; but treatment must at once be undertaken, to save the structure which is in trouble, whether it be intestine or testicle.

A boy, aged 1 year, suddenly developed a tense swelling about the size of an almond in the right inguinal region, and therewith had pain, with signs of general malaise. The right testicle was absent from the scrotum, and the left one had only incompletely descended. There were three possibilities: (1) strangulated hernia; (2) acute hydrocele in the inguinal canal; and (3) torsion of a testicle. All these three circumstances were favoured by the presence of an inguinal testicle. The absence of persistent vomiting, the softness of the abdomen, and especially the passage of motions, were points against a strangulated hernia. Flatus was retained, but nothing definite could be inferred from this. The marked disturbance of the general condition showed that it could not be a mere hydrocele. Torsion of the testicle remained, therefore, as the most probable diagnosis, and this was confirmed by the operation, which was performed forthwith. The little patient repeated the whole procedure, a year later, on the other side.

The same considerations should guide us in the case of torsion of a testicle, which is in the *scrotum*. There is, of course, no emptiness of the scrotum, as a diagnostic sign in such a case.

There are some circumstances in which we may hesitate as to the diagnosis between a strangulated hernia and swollen inguinal or femoral glands. If there are any intestinal symptoms there cannot be any possible doubt, and it is incredible that any practitioner should order poultices to what is really a strangulated femoral hernia, in order "to ripen" the swelling. The following case will, however, show that both patient and practitioner may succumb to a series of misleading circumstances.

A female, aged 50, became ill with acute cholecystitis, and suffered from very painful swelling of the gall-bladder. The initial vomiting ceased and the cholecystitis subsided somewhat. But at the same time the doctor accidentally discovered a tender swelling in the left groin. No definite pedicle could be distinguished. As there was neither vomiting nor colic, and as the abdomen was soft, and as there was an erosion on the corresponding lip of the vulva, which suggested a glandular swelling, the patient was watched for a day. The absence of the passage of flatus determined the diagnosis of strangulated hernia after the lapse of this time—but it was too late. The patient died suddenly during the preparations for operation. The autopsy showed not only a severe suppurative cholecystitis but also a strangulated hernia, with gangrene at the constricting ring. The strangulation evidently took place during the cholecystitis as a result of the vomiting, but the patient was not intelligent enough to notice it. There was no vomiting or colic because the patient was under dietetic treatment owing to the cholecystitis, and therefore the intestine was almost empty. The erosion on the vulva also contributed its share towards the error.

(2) Is the Hernia Strangulated?

One of the most important pieces of evidence for strangulation is afforded by the pain on pressure over the site of the constriction, usually at the hernial ring. The obstruction of the bowel, and the symptoms consequent thereon, constitute further evidence. This obstruction also occasionally occurs in Littré's hernia, which involves only the intestinal wall. Strangulation of omentum is distinguished from ordinary irreducibility thereof by the sudden onset of symptoms, the pain on pressure over the site of strangulation, and the tension of the hernia owing to the development of hernial fluid.

We have mentioned an example of such strangulation of omentum in the chapter on Inguinal Herniæ.

There is *one* further possibility in connection with strangulated hernia. Formerly one heard much about *inflammation* of a hernia, and strangulation was regarded as inflammation. There is nothing surprising in the fact that the sac of a strangulated hernia may inflame after a time, in consequence of infection by organisms which wander through from the bowel. The inflammation is, however, secondary in such circumstances. Primary inflammation of a hernia is, on the other hand, not frequent. The following conditions are most important:—

(*a*) *Appendicitis in a Hernial Sac.*—The appendix is not infrequently found in right-sided herniæ; it has even been found on the left side and also in umbilical herniæ. It may become inflamed within the hernial sac and perforate there. Such a case is usually mistaken for a strangulated hernia, until the error is rectified at the operation. But the sequence of symptoms ought to give a correct clue. In a strangulated hernia the first symptoms are those of intestinal obstruction, and then, after they have persisted for some time, comes the inflammation—the hernial phlegmon. But if appendicitis starts in a hernial sac, the inflammatory symptoms come first, and there is pyrexia. Should intestinal obstruction eventually supervene, it is a much later phenomenon. In most cases of strangulated hernia the pain on pressure is especially marked over the hernial ring; in appendicitis, however, it is from the first in the hernia itself.

(*b*) The involvement of the hernial sac in a *General Peritonitis.*—The differential diagnosis depends upon those considerations which were advanced in distinguishing between peritonitis and intestinal obstruction. The same applies to a hernia involved in the sequelæ of an *acute pancreatitis.* In both cases the severity of the abdominal symptoms will be very striking, considering the short duration of the supposed strangulation.

(*c*) *Tubercle of the Hernial Sac.*—This is usually a consequence of general tubercular peritonitis. But it sometimes happens that this latter causes no symptoms, and the practitioner is only consulted about the disease of the hernial sac. Whereas a miliary tuberculosis of the hernial sac with fluid contents is most likely to be taken for a hydrocele, the nodular form is easily confused with a strangulated, or at least with an irreducible, mass of omentum. If, however, one bears in mind the possibility of tubercle of the hernial sac, suspicion thereof will be aroused by the presence of isolated nodules, by the pain on pressure, and by the great hardness of the swelling. A careful examination of the abdomen may perhaps find support for the diagnosis. Abdominal pains are of less diagnostic significance, because, although they are frequent in tubercular peritonitis, they may also be caused by strangulated omentum.

(*d*) It has been shown that metastatic inflammation of an empty hernial sac may resemble the clinical symptoms of strangulation, but this is a rare condition.

There are three possible events which may introduce error into the solution of the last two problems, with which we have been dealing; first, the *combination of external hernia with internal intestinal obstruction.* Let us assume the case of a patient with all the signs of intestinal obstruction, in whom we also find an irreducible hernia, and we are inclined to ascribe to it the cause of the obstruction. If the hernial swelling is soft and not painful on pressure, the cause of the obstruction must be elsewhere; either in another hernia, which has been overlooked, or in some disease within the abdomen itself.

On the other hand, there are cases wherein we find no hernia, and are therefore inclined to attribute the intestinal obstruction to some internal cause. On inquiry we may learn that the patient has put back a hernia. Careful examination of the site occupied by the hernia may then show some slight retraction, and we may discover in the abdomen, behind the ring, an indefinite resistance, which is painful on pressure. Our diagnosis must then be that of *reduction en masse.* These cases are, however, becoming rarer, as fortunately violent taxis is giving way to herniotomy.

It occasionally happens that an omental hernia becomes painful on pressure. Symptoms occur, at the same time, suggestive either of peritonitis or of intestinal obstruction. There may be found in the abdomen a tumour-like painful area of resistance with corresponding dulness, or even, under some circumstances, a free fluid effusion. If these symptoms occur on the right side, in a patient who has already suffered from attacks of pain in this region, the diagnosis usually made is that of appendicitis with extension of the inflammation into the sac. This diagnosis seems all the more likely as the process is often accompanied by moderate fever. Operation, however, shows that the case is one of *torsion of a large mass of omentum*, the extremity of which is firmly fixed in the hernial sac—a condition with which all surgeons are familiar.

(3) What does the Hernia Contain?

If signs of intestinal obstruction are present, we must assume that the *hernia is intestinal;* if they are not present the hernia must be *omental*. One must be very cautious in drawing any conclusion from physical examination. A tympanitic note of course indicates an intestinal hernia. A dull note is of no significance, as small intestinal herniæ are very liable to yield complete dulness on percussion. The feel of the hernia can never be relied on. The granular consistence of omentum may be concealed by hernial fluid, and, on the other hand, a small coil of intestine is often found strangulated in cases wherein an omental mass can be definitely demonstrated. If there is a small movable body in the hernial sac of a female, the case is one of a *strangulated ovary*, a condition which is not rare, especially in young girls.

(4) Where is the Strangulation Situated?

The strangulation may be in the neck of the sac, at the hernial ring, or in the sac itself.

The strangulation is especially at the neck of the sac in inguinal herniæ, which usually present a ring-shaped thickening at the level of the internal ring. As this ring may shift as far as the peripheral end of the sac, in consequence of the constant onward movement of the peritoneum, the strangulation may occasionally occur quite close to the head of the sac. We may assume the presence of this condition, if the central portion of the hernial swelling is soft and painless, while the peripheral portion is tense, and tender to pressure. As a rule, a strangulation within the sac is at the level of the internal inguinal ring, and one may assert that most strangulations at this spot are strangulations within the hernial sac, because the internal ring is not itself tight enough to cause a strangulation. If the diagnosis is at all possible, it will depend upon the localization of the greatest amount of pain on pressure. Strangulation at the external ring is recognized in a similar manner, but here the strangulation is not caused so much by sac as by the fibrous elements of the inguinal ring.

In cases of *femoral hernia*, the strangulation usually takes place at the femoral ring; in *umbilical hernia*, it is due to constriction at the umbilical ring, but it may occur in one of the frequent pouches of the hernial sac. In the latter case, only one segment of the hernial swelling will be tense and painful on pressure.

Finally, there are cases wherein the intestine is caught by a noose-like band of connective tissue arising from the hernial sac. Such a condition is first recognized at the operation.

(5) What is the Condition of the Strangulated Gut?

The duration of the strangulation affords important information, because the intestine usually retains its vitality for the first twenty-four hours, though the constricting furrow may become necrotic after twelve hours. If, however, the constriction is not very tight, the intestine may recover itself, even after several days' strangulation. All depends upon the degree of circulatory disturbance, which is usually greater in small herniæ than in large ones. Gangrene is therefore more probable in a small hernial swelling than when a large mass of intestine and omentum is present. The presence of omentum in the hernia permits us to make a more favourable prognosis in regard to the intestine, because the omentum serves as a protecting pillow to it in the hernial ring. As long as the hernial swelling remains movable and the skin above it can be picked up, and is neither red nor œdematous, so long is the recovery of the intestine not impossible. But if inflammatory symptoms have occurred in any degree, from simple œdema of the integument to a definite hernial phlegmon, we must expect that the intestine has been severely damaged.

Although the indications are for immediate operation in all cases, nevertheless if circumstances prevent it being carried out forthwith, we may venture upon a modest attempt at taxis, if the strangulation is recent and inflammatory symptoms are absent. But if the operation can be done at once—and this is always the case nowadays, except in remote districts—one may quite conscientiously abandon taxis. Infants, however, provide an exception. Strangulated hernia is very rare during the first year of life; at this period herniæ are easily reducible and never cause gangrene. As a rule, the herniæ goes back when the child is put in a bath.

(6) The Questions which may Arise during the Operation.

We shall not waste any words over the recognition of the various layers of the hernial coverings—a hobby-horse of the older surgeons. The layers may represent the usual coverings in recent herniæ, but in old herniæ they may be considerably increased in number by the growth of new layers of connective tissue. The careful operator will succeed in entering the hernal sac, *tuto* if not *cito*, even without counting the layers; but he must remember that not every space containing fluid is the hernial sac. Cystic spaces containing serous fluid often occur around the hernial sac, especially in femoral hernia, and if the hernia is strangulated the serous fluid may be mixed with blood.

The decision made from the *appearance of the intestine* is of greater importance; and it is necessary to consider not only the strangulated coil but also the bowel leading towards it. In order to pull it sufficiently forward it is necessary to widen the constriction previously, but care must be taken lest the hernial coil slips back into the abdomen unexpectedly and unperceived. If the intestine is smooth and shiny, and if definite contractibility is present in the whole of the strangulated coil, including the constricted ring, we may safely reduce it, even though it may have seemed congested at first and felt somewhat thick. The bluish discoloration improves as we wait, and the thickening is preferable to the opposite condition. The intestine may be suspected if the contractions only start after long waiting, and are then very indolent. In such doubtful cases the circulation in the mesentery must be observed, and special attention must be paid to the arteries to note whether they are pulsating, and to the veins, to see whether they are thrombosed. Intestine should not be replaced if it cannot be excited to contract in every part, including even the constricted ring. Even if its consistence remains normal, or only slightly thickened, it should not be replaced. It is quite certain that if necrosis has once started it will become complete, in cases wherein the intestinal wall is disposed in small folds and its consistence is diminished, whether its colour be black, green, or grey. The character of the hernal fluid may prove of value in doubtful cases. A clear, odourless fluid indicates that the intestine is capable of recovery; a turbid offensive fluid shows that necrosis has begun. Obviously, we must not allow an apparently healthy hernial fluid to reassure us, if, for instance, we find clear signs of necrosis at a constricting ring. On the other hand, the fluid may be somewhat offensive or slightly turbid in cases wherein the bowel is capable of recovery.

I was once called to a distant village to see an old woman with a strangulated femoral hernia. When I arrived the practitioner informed me that the symptoms of obstruction had subsided, but that the hernial swelling was increasing. The swelling felt very tense, and when it was opened a little offensive gas escaped. This led into the femoral hernial sac, which contained a very few drops of pus, and which was separated from the abdominal cavity by obviously recent adhesions. Recovery followed in a short time without further intestinal disturbances. The intestine which had been strangulated had returned of its own accord, leaving behind some bacteria, which did nothing more but infect the hernial sac. This case reminds us of the observations which we have already made in regard to inflammation of an empty hernial sac.

(7) Questions which Arise after Reduction by the Open or Bloodless Method.

A coil of intestine which has been strangulated for some hours does not always resume its functional activity forthwith. We must, therefore, not be surprised if some colic still persists for several hours, or for a day or two after the strangulation has been relieved. It may not be easy to obtain the passage of motions and flatus even after copious enemata. If reduction has been effected by taxis we must not allow this fact to reassure us very easily, but should think of the possibility of a *reduction en masse.* An operation must be resorted to if the symptoms continue for several hours with the same severity. If herniotomy has been performed we may wait longer, but must undertake laparotomy if the symptoms increase instead of abating, especially if the pulse becomes unsatisfactory. There may be some additional intra-abdominal trouble, such as a volvulus, or kinking, owing to adhesions. The acceleration of the pulse should suggest to us that we have probably erred in deciding that the intestinal coil retained its vitality.

I have seen gangrene come on in an entire coil after a skilfully performed herniotomy in a case wherein the strangulation had not existed for twelve hours.

There are some cases in which everything goes well at first, but wherein the patient begins to complain again, after the lapse of several weeks, of attacks of colic, and finally, of retentions of motions, and flatus—in a word, of the symptoms of chronic intestinal obstruction. If we operate we find that a ring-shaped or a channel-shaped narrowing exists in the place of the strangulated coil, or that the whole coil has been caked into an inextricable ball through adhesions. Both these conditions show that we were too optimistic in our opinion of the intestine at the time of operation. They both indicate that the mucous membrane has sloughed more or less extensively, and this may occur even when the serous surface appeared to be capable of living and has, as a matter of fact, retained its vitality. The only early warning of the onset of this *late stenosis* is offered by intestinal hæmorrhage and persistent diarrhœa coming on in the first weeks after the reduction.

A female, aged 60, was operated on for strangulation of an umbilical hernia, which had persisted for eighteen hours. The intestine appeared to be healthy and was replaced. The intestinal functions were normal for the first few days, but classical symptoms of chronic intestinal obstruction occurred after the second or third week. They increased so much that another operation became necessary in the seventh week. The intestine which had been strangulated had developed into an S-shaped mass, with very firm adhesions of one portion of the coil. An entero-anastamosis was performed, with complete and permanent recovery.

CHAPTER LVI.

DIFFICULTIES OF DEFÆCATION.

In the popular sense this term includes several conditions of varied significance which the practitioner must separate from one another. They comprise functional derangements of the whole intestine, or, at least, of the large intestine, and also disorders which are due to some local affection of the rectum or its vicinity. We have discussed the former in the section on "intestinal obstruction."

The following are the symptoms by which rectal disease manifests itself :—

(1) *Simple Constipation*, i.e., *Difficulty in evacuating the Fæces, but no Pain or Tenesmus.*—This occurs in simple weakness of the rectal muscle (proctogenic constipation), or with tumours of the true pelvis which mechanically compress the rectum from without, and a fair amount of compression is required before defæcation is compromised. **Retroflexion of the gravid or myomatous uterus, exudations within the true pelvis, cysts, and solid tumours** may be mentioned as examples. The notorious "tape-like" fæces are sometimes found in these circumstances.

We have been told by a patient with a dermoid in the pelvis that his motions were as thin as cardboard.

(2) *Simple tenesmus* without any disturbance of evacuation and without any hæmorrhage. This generally indicates a tumour pressing upon the rectum, without entirely obliterating its lumen, e.g., a very large prostate, retro-prostatic cysts and also malignant growths of the pelvis at the initial stage of their development.

(3) *Simple hæmorrhage* without any other disturbance. If the blood is not mixed up with the fæces but merely covers them over, or if the blood appears independently of defæcation, it indicates disease of the lower part of the rectum. If the blood is mixed with much mucus, some form of colitis is indicated. Pure blood is

occasionally seen in cases of cancer, especially if they are situated high up, but it occurs more frequently in cases of polypi and hæmorrhoids. A blood-stained serous discharge usually indicates cancer, either of the ampulla or the anus. Any hæmorrhage should suggest the necessity of a very careful examination. It is very easy to overlook a bleeding polypus, or a bleeding carcinoma, and ascribe the symptoms to ordinary hæmorrhoids.

(4) *Constipation associated with Constant Tenesmus.*—The evacuations are soft and pulpy, or even quite liquid, and are frequently expelled in very small quantities at a time. The patient experiences an incessant desire to go to stool. This picture is most frequently encountered in cases of progressive pressure on the rectum from without by one of the swellings just mentioned.

(5) *Tenesmus, without Constipation, but with the Passage of Blood, Mucus or Sero-sanious Fluid, during the Acts of Defæcation, and in their intervals.*—If a patient complains of these symptoms, and especially if his clean shirt is soiled with red or pinkish stains, we may at once assume that some ulcerative process is present, which does not lead to stenosis. It may be associated with an ulcerative colitis, extending low down, an innocent polypus, a syphilitic or tuberular lesion, or it may be due to cancer which has not yet reached the stage of stenosis—the most frequent cause.

(6) *Tenesmus, with Constipation, Passage of Liquid, Pulpy or softly formed Fæces in small Quantities, accompanied by Blood or Sero-sanious Fluid.*—This assemblage of symptoms points to an *ulcerative* lesion, which is also *causing stenosis,* i.e., to cancerous, syphilitic, or gonorrhœal stricture.

The history affords certain indications for the exact diagnosis of the above mentioned conditions; thus in childhood polypus is the most frequent cause; in females from adolescence to middle age syphilis or gonorrhœa is, under certain conditions, the most probable cause; in elderly people a villous polypus, or cancer, is most likely to be present. Examination of the rectum must never be omitted. The trouble must never be attributed to piles, which may accidentally be present, and they should never serve as an excuse for an incomplete examination. That so many advanced cases of cancer are sent for operation with the mistaken diagnosis of rectal catarrh or hæmorrhoids is due to the shyness of the patients—and often of practitioners—in the matter of rectal examination. It is quite intelligible and praiseworthy that the practitioner who is engaged in surgery or in obstetrics should avoid contaminating his fingers unnecessarily; but nowadays a finger-stall removes the risk of contamination, and a rectal examination in this type of case is never "unnecessary."

If we feel a soft movable structure, with a more or less long stalk, which easily escapes the finger, the case is one of **polypus**—a *mucous polypus* in children, a mucous polypus or a *villous polypus* in adults. If the mucous membrane appears to be softly granular over a diffuse area, or if it be studded with superficial ulcers, which are most distinctly recognizable with a rectal speculum, the condition represents an early stage of chronic **gonorrhœal or syphilitic proctitis.**

Flat, very painful ulcers in the vicinity of the anus suggest **tubercle.** We have already discussed the differential diagnosis between this condition and non-specific ulcerative colitis.

If the finger defines a shell-like elevation of the mucous membrane with an overhanging, more or less firm border, and a friable, easily bleeding surface, the case is one of **cancer,** which has not yet encircled the bowel. If there be any doubt, such as might arise in the presence of a flat non-pedunculated papilloma, a histological examination must be made. Such papillomata often become malignant. The same applies to the **polypoid condition of the rectum,** in which the whole rectum, and often a considerable portion of the large intestine, is invaded by numerous polypi. If the finger enters into a smooth-walled, rigid, cylindrical tube, the case is one of **stricture,** following **gonorrhœal** or **syphilitic proctitis.** If, on the other hand, we feel a circular wall, into whose centre the finger can scarcely impinge, owing to the narrowness of the opening, and if the friable tissue breaks down, there can be no question that we are dealing with a **ring-shaped carcinoma.** If the growth is high

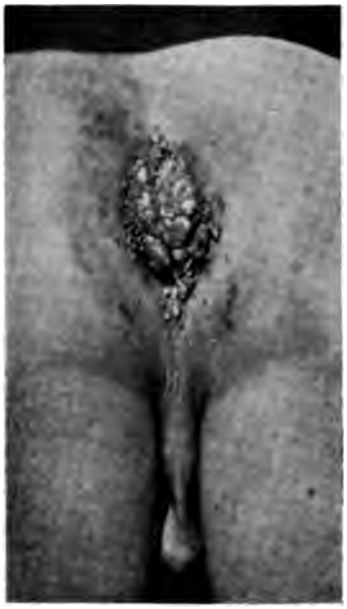


FIG. 238. — Acuminate condyloma of anus.

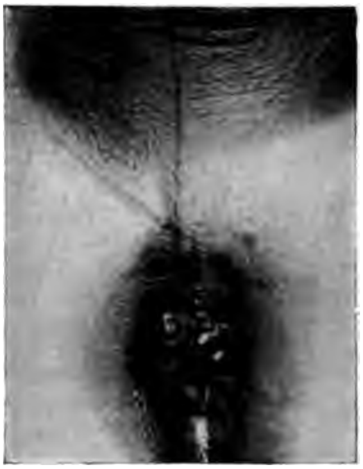


FIG. 239.—Relaxed hæmorrhoids.

up, we must direct the patient to bear down, or we must press the abdomen downwards with our free hand. According to Hochenegg, a remarkably wide ampulla should raise the suspicion of a cancer, situated high up, and fixing the pelvic colon. If a cancer of the lower segment of the rectum protrudes through the anus the diagnosis is easy enough (fig. 242). **Sarcoma** is much more rare in this region (fig. 245).

We also append, for the sake of comparison, an illustration of acuminate condyloma (fig. 238). The diagnosis is easy, even if no information about its etiology be forthcoming.

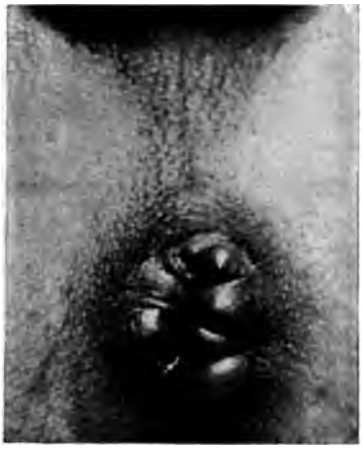


FIG. 240.—Inflamed external hæmorrhoids.

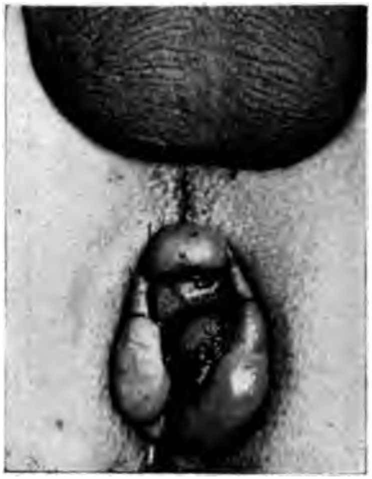


FIG. 241.—Prolapsed internal hæmorrhoids surrounded by external œdematous piles.

(5) *Painful Defæcation.*—We meet with this symptom in various conditions :—

(*a*) If a patient tells us that he suffers from severe cutting pain in the anus after every action of the bowels, that the pain starts immediately, and lasts for about fifteen minutes or longer, and that he postpones defæcation as long as possible on account of the pain, we may conclude that he is suffering from a **fissure**. On examining the anal aperture, we see one or more radiating cracks which, if carefully separated from each other, look like defects in superficial epidermis, with a reddened base. Sometimes they are situated between small nodules, of hæmorrhoids of a perfectly unirritating character.

(*b*) If, on the other hand, the pain is only occasional, and comes on in attacks lasting for a few days, the pain at first being confined to the moment of defæcation, but subsequently becoming more persistent, only to disappear for some time, after the loss of considerable dark blood, we may be certain that the condition is one of inflammatory changes in **hæmorrhoids,** i.e., thrombosis in their venous spaces. One of my patients compared the severity of the pain to toothache. Sometimes the loss of blood only occurs periodically at long intervals, at other times the loss of blood may be constant at each action of the bowels over a protracted period—the loss being to the extent of a dessertspoonful or more. The condition found on inspection varies with the stage in which the patient happens to be. We may only find a few withered folds of skin or mucous membrane (fig. 239), or a bunch of bluish-red tense nodules, tender on pressure, or even a whole crown of them. If the patient is in the bleeding stage, one of these nodules will be ulcerated, and it may be possible to see a black coagulum projecting from the point whence the hæmorrhage occurred. If internal hæmorrhoids became thrombosed, they prolapse very easily, and may be strangulated by the sphincter ani, with such damage to their circulation that necrosis may follow. They look like bluish-black or brownish-black nodules, surrounded by œdematous anal skin, or by a ring of œdematous and swollen external hæmorrhoids (fig. 241). Further extension of the inflammation may cause retention of the urine, abscesses, and even general septic infection.

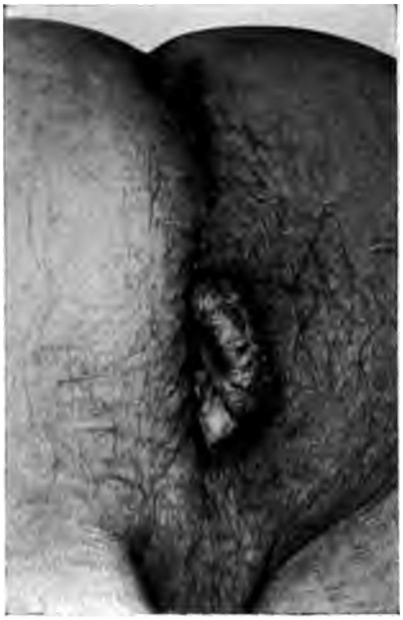


FIG. 242.—Cancer of the anal portion of the rectum.

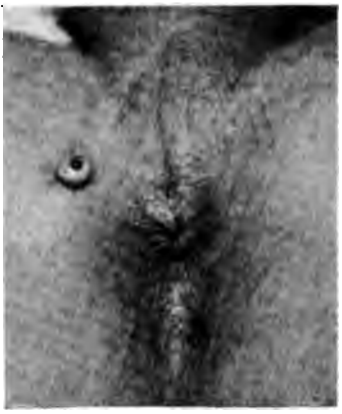


FIG. 243.—Rectal fistula.

If the subjective symptoms and the hæmorrhage indicate the one of **prostatitis**, probably of **gonorrhœal** origin, but possibly also of a **tubercular** nature.

(*e*) If the symptoms do not fit in with any clinical picture, but consist of tenesmus, blood and possibly pus in the stools, constipation, pains in the pelvis, we should think of a **foreign body**. We shall see in the next chapter, the variety of articles which have been introduced into the rectum.

(6) *Prolapse of the Anus.*—Sometimes the difficulty in defæcation consists of a prolapse of the rectal mucous membrane. The diagnosis can be made by a layman; we have to decide whether the prolapse merely concerns the lowest portion of the mucous membrane—**prolapsus ani**—or whether the mucous membrane higher up is involved—**prolapsus recti**—or whether both conditions are present (fig. 244). Our decision must be based on the size of the prolapse and upon the level at which the fold in the bowel is found—at the anus or higher.

Prolapsus ani is purely a matter of mucous membrane prolapse. In *prolapsus recti*, all the layers of the intestinal wall are turned in and the peritoneum may even be dragged, so that a perineal hernia may be formed—a so-called hedrocele.

CHAPTER LVII.

INJURIES OF THE RECTUM.

APART from lacerations of the intestine and rectum during labour, the injuries of this region are mostly due to impalement or to foreign bodies introduced for various purposes and in different ways.

Impalement may be caused by falling on a garden fence, the handle of a broom, the barrel of a gun, or the leg of a chair, &c. Agricultural labourers are often impaled by falling off a haystack on to the handle of a fork. The laceration of the rectum by the horns of a bull produces a similar injury to impalement. The external wound is easily recognized in all these accidents; diagnosis is mainly concerned with the question of the extent of the injury above. This may sometimes be determined by the length of the tract of blood on the impaling instrument.

Some consideration should be bestowed upon the question of the possibility of a foreign body being retained in the wound. The following case, which I saw when assistant to Kocher, shows that this is not an unimportant matter. A young man, overstimulated by alcohol, climbed up to the top of three chairs standing on each other. The chairs fell over, and the leg of one of them penetrated deeply between his rectum and sacrum. But the wound seemed to be remarkably harmless, and the rectum appeared to be uninjured. The onset of fever and of offensive discharge showed, however, that there was something wrong. Just before the demonstration of the patient in the clinic the probe touched at the level of the promontory on a peculiar structure, the meaning of which at once became clear. During the clinical demonstration the seat of the trousers in which the four seams joined together was triumphantly extracted by the forceps. The patient had changed his trousers before being brought to the hospital, otherwise the hole would have enabled us to make the diagnosis earlier.

If we are certain from the length of the penetrating body that the peritoneum has been torn, the abdomen must be opened and searched, so as to avoid a belated diagnosis of intraperitoneal injury to the intestine, when peritonitis supervenes after a few days.

Among the **foreign bodies purposely introduced**, one should mention especially the *nozzles of enema syringes*. Fatal injuries have been inflicted in this way, especially when the enema is administered into the abdominal cavity. These accidents happen, naturally, more frequently when the mucous membrane is diseased, as in cancer, than when it is healthy.

In contrast to an enema syringe employed for constipation, a wood-chopper in the forest put a wedge of wood into his rectum, for diarrhœa. A colleague has informed me of a case wherein a man introduced a liqueur glass, in order that he might be able to witness a festive procession, undisturbed. Sometimes foreign bodies are used for masturbation.

There is no complete account of these cases in literature, and the most unique examples of these occurrences do not exhaust all the possibilities, but in them all the patient complains of tenesmus and hæmorrhage. We should, therefore, never neglect a rectal examination in any case wherein such symptoms come on, apparently without cause.

As we cannot obtain any reliable information concerning the nature of the foreign body, we must be careful to protect the finger with a stall, to prevent injury to it. Inspection with a speculum, which is sometimes indispensable for the diagnosis, should be left until the extraction is undertaken, as this requires anæsthesia—general or local.

The rectum is occasionally injured during attempts to procure abortion. I once made an autopsy on a young girl who died of peritonitis after an attempt at abortion. The posterior vaginal wall and Douglas' pouch were perforated and the rectum was impaled: The midwife who was accused set up the defence that she was much too experienced to have attempted to gain the right orifice in this clumsy manner.

CHAPTER LVIII.

TUMOURS AND SWELLINGS OF THE SCROTUM.

WHEN we are consulted for any scrotal swelling, we must first determine whether the swelling originates in the testicles or in the scrotum. This can only be done if it is possible to feel the testicle and its appendages quite distinctly, apart from the investing skin. But if the swelling, the testicle and the skin all form one connected mass, we must depend upon the history to tell us whether the change involved at first the scrotal contents only.

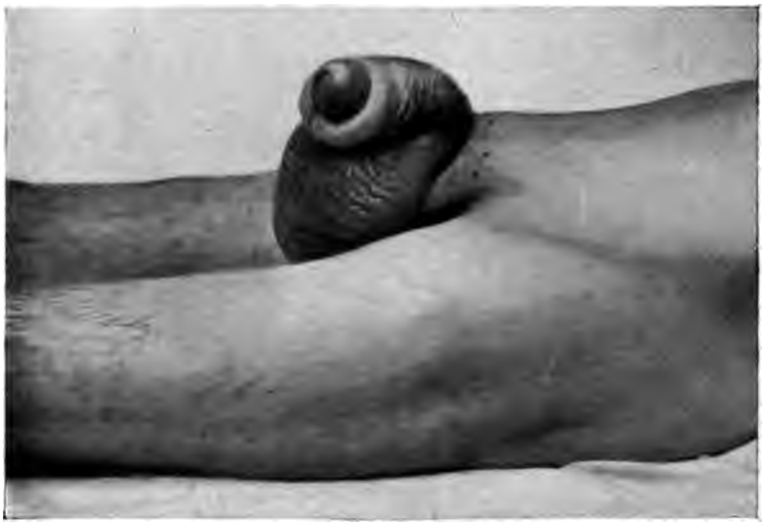


FIG. 246.—Acute extravasation of urine, in a case of neglected urethral stricture.

I.—SWELLINGS OF THE SCROTUM.

A.—ACUTE SWELLINGS.

If the swelling has arisen suddenly, the first point to consider is the question of **contusion**, which may cause a severe degree of blood extravasation. Then there is the possibility of **acute inflammatory diseases**, especially erysipelas, or of **extravasation of urine** consequent upon an injury or a neglected urethral stricture, a matter of the greatest importance, because the treatment depends upon the accuracy of the diagnosis. If one meets with such a clinical picture as is illustrated in fig. 246, he must not be content with the diagnosis of phlegmon, &c., but must at once provide for the free escape of the urine, in order to prevent his patient dying from uræmia and sepsis.

B.—CHRONIC SWELLINGS.

(1) If a diffuse swelling has arisen gradually, as a result of repeated acute inflammatory attacks, e.g., erysipelas, or as a consequence of chronic multiple urinary fistulæ, the term applied to it is **elephantiasis.** The same name is applied to the enormous hypertrophy, which occurs in the tropics, from filarial disease. In this condition the patient is hardly able to walk, because of the appendage to his scrotum.

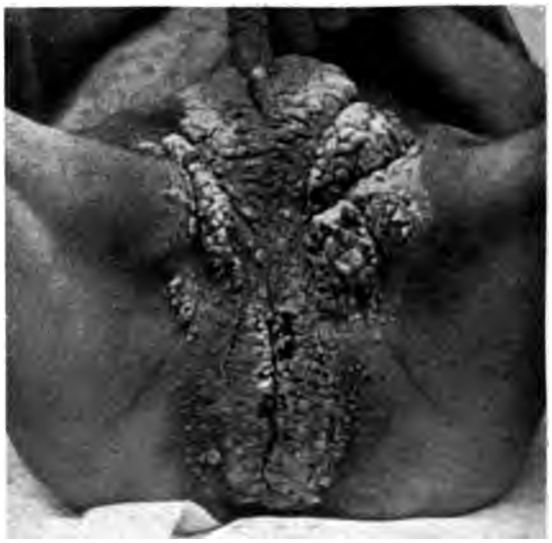


FIG. 247.—Psorospermosis of the perinæum.

(2) *Circumscribed tumours* of the scrotal skin are diagnosed according to their consistence. If they show fluctuation, are soft or tensely elastic, they may be **dermoids, sebaceous cysts** or **cystic lymphangiomata.** The former are unilocular and non-translucent; the last are multilocular and translucent, but are distinguished from hydrocele by their superficial situation.

If the circumscribed tumour is hard, it may be either a **fibroma** or a **sarcoma,** in accordance with its rate of growth.

Ulcers on the scrotum are usually primary chancres, but occasionally are of a gummatous nature, and in rare instances may be tubercular. This last variety is illustrated in fig. 248. Its appearance exactly resembles that of a hard chancre. If the patient's occupation is concerned with soot, tar or paraffin, and his ulcer has been preceded by an eczematous condition, we may assume that it is a case of chimney-sweep's or paraffin cancer—an assumption which should be confirmed by histological examination. Broad condylomata are easily recognized, owing to their multiplicity, even under their most grotesque forms (fig. 250). Psorospermosis of the scrotum is a very rare condition (fig. 247).

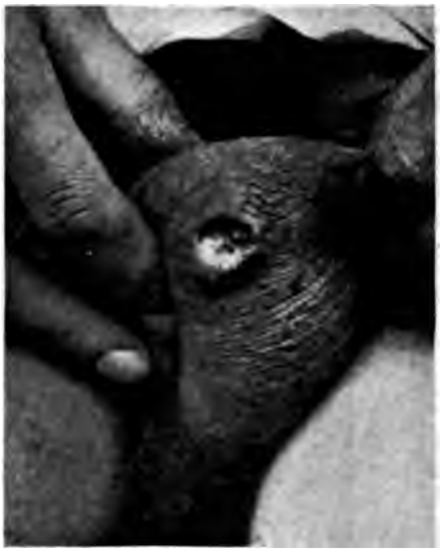


FIG. 248.—Tuberculosis of scrotum.

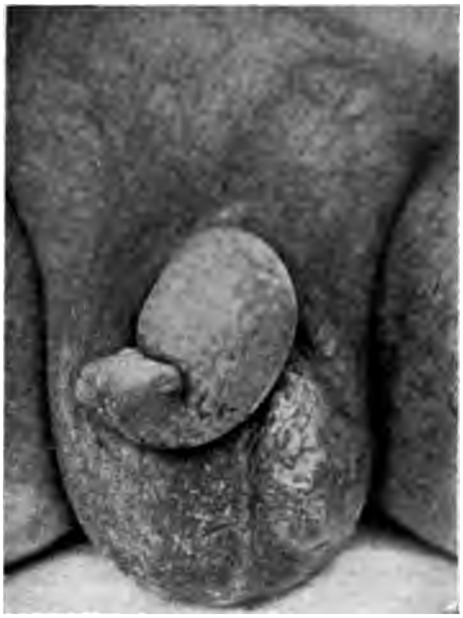


FIG. 249.—Elephantiasis of penis and scrotum, with an indrawn urinary fistula on the left. (Case of neglected stricture).

II.—SWELLINGS OF THE SCROTAL CONTENTS.

If the swelling is in relation with the normal contents of the scrotum, we must first ascertain whether it has a pedicle at its upper part, in other words, whether it runs into the inguinal canal. If this should be the case, the conclusions to be drawn are already discussed in the chapter on Inguinal Herniæ.

In acute inflammation of the testicle and epididymis there is nearly always some infiltration of the spermatic cord, which may then resemble the pedicle of a tumour, a matter which will be referred to later on. The same applies to advanced cancer.

A.—TUMOURS OF THE SPERMATIC CORD.

An elastic or tense swelling in connection with the spermatic cord, quite free of the testicle and epididymis, is a **funicular hydrocele.**

The diagnosis is absolutely certain if the swelling is, in addition translucent.

We have seen in Chapter LV. that the acute hydrocele of little boys is often mistaken for strangulated hernia.

A soft non-translucent swelling is in all probability a **lipoma of the spermatic cord.** A hard tumour, adherent within the spermatic cord, suggests the rare form of **sarcoma,** which grows rapidly towards the abdominal cavity.

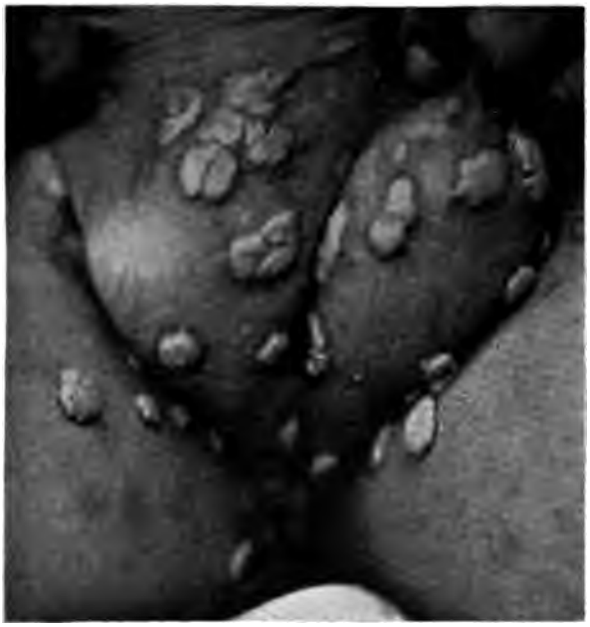


FIG. 250.—Broad condylomata of the scrotum.

We now proceed to deal with diseases of the testicle and epididymis, which, logically, should be considered separately. But as the apparent position of the swelling does not always correspond with its true topography, we propose to group them together in the first instance and then, later on, endeavour to distinguish between the testicle and epididymis.

B.—ACUTE SWELLINGS OF THE TESTICLE AND EPIDIDYMIS.

Let us assume the case of a patient who consults us for an acute painful swelling of the scrotal contents, and who tells us that it has resulted from a blow, a statement which should be taken *cum grano salis* if the patient is insured against accidents. We find the testicle and epididymis fused together into a very tender oval-shaped mass. The skin of the scrotum is slightly œdematous, the spermatic cord is hard and swollen, and resembles a pedicle running into the inguinal canal, and the vas deferens cannot be felt as a separate structure. If we agree that an injury has been the cause, we should think of a **traumatic hæmatocele,** i.e., an effusion of blood into the tunica vaginalis and into the cellular tissue of the scrotum. The diffuse swelling which obscures the division between testicle and epididymis and the infiltration of the spermatic cord may very well be attributed to the œdema which accompanies such an injury. Our assumption will be correct if the swelling exceeds in size the usual dimensions of a recent orchitis or epididymitis, i.e., if it is larger than a goose's egg. Sometimes confirmation may be obtained from a dark blue discoloration of the scrotum, especially on the posterior surface; at any rate this discoloration should appear within a few days. If, however, the swelling be smaller, and there be no discoloration despite the lapse of a few days since the alleged accident, our thoughts should run in another direction, although we should not entirely discard the traumatic theory. Pressure on the urethra may cause a few drops of the anticipated discharge to exude, and then further discussion is unnecessary, especially if the swelling mainly concerns the epididymis. The case is one of **gonorrhœal epididymitis.** If we do not obtain any discharge, which sometimes ceases with the onset of the epididymitis, we should inquire for the date of the last attack of gonorrhœa. If this meets with a negative reply, we should make a rectal examination, exerting slight pressure on the prostate, and then direct the patient to urinate. Abundant gonorrhœal threads and small flakes of pus suffice for the diagnosis. But if these are not present, and the urine is somewhat turbid and of offensive odour, containing numerous pus-cells, micro-organisms, and probably crystals of triple-phosphate, we must abandon the idea of gonorrhœal epididymitis. The patient has cystitis either as the result of stricture, hypertrophied prostate or tubercle, &c., and the **epididymitis** or **orchitis** has arisen owing to extension from the bladder. As these infections are frequently caused by trifling injuries, we should ascertain whether the patient has had a catheter passed shortly before the onset of the swelling.

All this does not exclude the possibility that gonorrhœa may have been the original starting point of the disease. The gonorrhœa may have occurred years previously, and the infecting organism at the moment is not necessarily the gonococcus, but an ordinary pus organism such as the staphylococcus or streptococcus.

We may assume inflammation of this kind if the testicle rather than the epididymis is involved. But if no source of infection is discoverable in the uro-genital apparatus there remains the possibility of **metastatic orchitis.** This includes the inflammation of the testicle which sometimes occurs in mumps, and occasionally after other infectious diseases such as typhoid fever. Finally, there are some very rare cases which cannot be explained, even in this manner; the orchitis is apparently spontaneous and has no connection with any other disease. The testicle is, as a rule, alone involved in these cases, so that they are easily distinguished from gonorrhœal inflammation.

If this form of swelling appears to extend beyond the testicle, it usually depends upon a slight attack of secondary hydrocele, in association therewith. In such a case there would be no sharp separation between testicle and epididymis; but, as stated previously, they would emerge into one oval-shaped swelling. In a simple orchitis the epididymis is situated above the swollen testicle like a narrow ledge. On the other hand, in a case of simple epididymitis, the uniformly enlarged epididymis lies against the testicle like the crest on a helmet.

If we are in doubt about the nature of an orchitic swelling owing to the absence of any accurate history, we may resort to an exploratory puncture, lest we overlook a purulent inflammation until it is too late to save the testicle.

Before making the puncture we should warn the patient that his testicle may atrophy as the result of the disease, otherwise the incident which happened to us, owing to our neglect of this precaution, might be repeated. A young man had a testicle as large as a plum, which was very painful on pressure; fever and pain persisted so that a puncture was made with a fine needle, but without any result. The inflammation, however, subsided, and the patient was discharged. He returned in a few days looking very sad, because not only had the inflammation disappeared but the testicle also, and he blamed the exploratory puncture for this. Indeed, there only remained a hard body, not bigger than a bean, instead of the plum-sized swelling. We had then to offer the explanation which we had previously neglected to do by way of prognosis, and the patient finally consoled himself with the reflection that the presence of one testicle was sufficient.

It happens occasionally that, although everything points to a gonorrhœal epididymitis, we learn that the patient has had of late more frequent calls to micturition than usual, the stream still being normal, but that he is obliged to get up one or more times during the night for this purpose. He may have noticed a fine, whitish deposit, or, exceptionally, a little blood in the urine. This usually signifies the beginning of uro-genital tuberculosis. How, then, is the acute onset of the swelling to be explained? This may be due to an acute perforation of a tubercular focus into the tunica vaginalis, with a consequent tubercular hydrocele. Examination would show that the epididymis cannot be separately defined fron the testicle, and that the latter appears to be enlarged, and may even present fluctuation. Or it may be that the epididymitis is not purely of a tubercular nature. There may have been a nodule which the patient had not noticed, and it may have become secondarily infected, a circumstance which often happens in uro-genital tuberculosis, even if catheterization has not been practised. Examination of the urinary sediment shows mononuclear and polynuclear leucocytes, possibly a few red cells also, epithelial cells, *Bacillus coli*, staphylococci and streptococci. As the disease progresses an abscess will probably form in the epididymis, and spontaneous rupture finally occurs.

Contusion of the testicle and the various forms of inflammation do not, however, exhaust all the possible causes of acute swelling. If the symptoms have come on very suddenly, and are accompanied by such reflex signs as vomiting, retention of flatus, if the swelling is somewhat high up and we are informed by the patient that his testicle had never completely descended, we should think of **torsion of the testicle,** a condition already referred to in connection with strangulated hernia. The results of this torsion are hæmorrhagic infarction and gangrene of the testicle.

The anatomical basis of this event is an abnormally developed mesentery of the testicle, which permits it to hang free in the tunica vaginalis. The only condition with which it could possibly be mistaken is an *embolic* infarction of the testicle occurring in a patient with heart disease.

C.—CHRONIC SWELLINGS OF THE TESTICLE AND EPIDIDYMIS.

Although we are not able to distinguish the testicle clearly from the epididymis when they are acutely swollen, the distinction is somewhat easier in the early stages of chronic swellings.

(1) Swellings of the Epididymis.

If the epididymis is hard and swollen in an irregularly nodular manner, or if a hard, tender nodule is felt in an otherwise normal organ, we should immediately think of tuberculosis. We seek for confirmation of this diagnosis in the characteristic nodular or cylindrical thickening of the vas deferens, which is early recognized because the vascular elements of the cord usually feel quite normal when the tubercle is not accompanied by secondary infection, in contrast to what we have seen in acute orchitis, gonorrhœal epididymitis, and tubercle with secondary infection. If the vas deferens is not thickened, we must look for traces of tubercle in the prostate, bladder, kidneys, as described in fuller detail in the chapter on uro-genital tuberculosis. The kidney is often the organ first affected, and the disease of the epididymis is only detected first, because of its accessibility.

There are three conditions with which this early stage of tuberculous epididymitis may be confused :—

(*a*) The *remains of gonorrhœal epididymitis*, hard, somewhat tender, indurations in the epididymis. The distinction is made by the history and other physical findings, especially by a careful examination of the urine.

(*b*) *Syphilitic epididymitis of the secondary stage*, recognized by its almost painless onset and other diagnostic signs of syphilis.

(*c*) A small cyst connected with the head of the epididymis—**spermatocele.** Its striking mobility, the smoothness of its surface, and its painlessness settle the diagnosis.

But if we do not see the patient until a later stage of the disease, when he has an old, retracted, slightly discharging sinus, the problem is quite different. Testis and epididymis are fused together into one shapeless mass and cannot be felt separately. The principal question to decide is whether the case is one of tubercle or *tertiary syphilis*. A gumma attacks the testicle by preference, while tubercle starts in the epididymis. The patient may perhaps be able to inform us of the original site of the disease, but if he cannot do so we should diagnose a gumma when the epididymis is only slightly involved and the testicle considerably affected, especially if spontaneous pain and tenderness are but slight.

If a sinus has already formed it is convenient to remember Reclus' sign to the effect that a syphilitic sinus generally lies *in front* and that a tubercular sinus lies *behind*, corresponding to the situation of the two diseases in the testicle and epididymis respectively.

One must not forget, however, that if the testicle is inverted a tubercular sinus may lie forward. It is therefore necessary to observe the position of the vas deferens in the spermatic cord to see whether it is in front or behind, before drawing any conclusion from the situation of the sinus.

(2) Swellings between the Testicle and Epididymis.

Cystic tumours lying between the head of the epididymis and the testicle, and seated on the latter like a cap or helmet, are grouped together under the term **spermatocele.** When such a cyst is present it is either impossible to feel the head of the epididymis as a separate structure, or it lies, as just stated, on the spermatocele, so that the latter is fixed between it and the testicle. In order to render the diagnosis more certain an exploratory puncture may be performed, which, although generally superfluous, never does any harm if asepsis is preserved. The diagnosis is confirmed by the turbid watery appearance of the liquid, and by the presence of seminal threads visible under the microscope.

(3) Swellings of the Testicle.

(*a*) An oval or pear-shaped tumour with a smooth surface and of soft or tensely elastic consistence, indicates an accumulation of fluid in the tunica vaginalis. If it be of small extent, the epididymis can still be appreciated as a separate structure, and we may even be able to feel the testicle if the fluid is not very tense. If, however, the swelling is large and the tension greater, both testicle and epididymis only present somewhat more resistant places in the wall of the swelling. If the effusion has a thick wall, it may be quite impossible to define them. The case is one of **hydrocele or hæmatocele of the testis, a serous or proliferating or hæmorrhagic peri-orchitis,** which may vary in size from a hen's egg to enormous proportions, if the patient waits until it is necessary to draw off the fluid by the pint.

If the swelling is translucent it is a hydrocele, and puncture is then not merely an exploratory procedure, but is a therapeutic measure, at any rate of a palliative nature.

If puncture of a tense translucent swelling is not followed by a flow of fluid, we should not diagnose cancer as a young practitioner once did, but rather a bad syringe.

If, however, the swelling is not translucent, the diagnosis is usually proliferating peri-orchitis, i.e., a hydrocele whose wall has become thickened by connective tissue proliferation, by crest- and cone-like indurations, and by deposits of gradually organizing fibrin. If, on exploratory puncture, a fresh bloody fluid, or more frequently a chocolate-brown fluid, exudes, the case is one of hæmatocele, which we must look upon as a sub-variety of proliferating peri-orchitis, when it is not of traumatic origin.

Serous peri-orchitis, as well as the proliferating and hæmorrhagic forms, give rise, in different ways, to many points of differential diagnosis.

A *serous peri-orchitis,* associated with appreciable changes in the testicle and epididymis, may be *secondary* or *symptomatic.* Such effusions within the tunica vaginalis occur in herniæ, tubercular epididymitis, and cancer of the testicle; but they rarely become so large that the underlying disease is obscured. If we find that the upper end of a hydrocele which extends high up is very painful on pressure, and that there is probably some thickening of the spermatic cord at this spot, it is very likely that a *fragment of omentum has become* *strangulated* in a communicating sac with a narrow neck, so that the hydrocele is another instance of the secondary form.

In a case of *proliferating peri-orchitis,* our diagnostic reflections take another direction, at any rate before an exploratory puncture is done. A *malignant growth* will very frequently suggest itself ; unequal consistence usually indicates a tumour, although in simple proliferating peri-orchitis its wall may present soft thin areas as well as hard ones. A rough nodular surface is clear evidence of new growth. Some tumours, however, have a perfectly smooth surface in their initial stage. As the infallible signs of a malignant growth, i.e., enlargement of the retroperitoneal glands of the same side and other metastases, are absent at the beginning, we must fall back upon the history. A simple proliferating or hæmorrhagic peri-orchitis has usually existed for months, or even years, whereas a malignant tumour can only have been present at most for a few months.

There is nothing absolutely conclusive in these signs, because a hæmatocele may develop very rapidly after an injury, and some malignant tumours grow very slowly. I once saw a malignant tumour which had been under the observation of the family practitioner for many years. These are, however, exceptional cases, and the practical rule remains that the duration of hæmorrhagic peri-orchitis is a matter of years, while that of cancer or sarcoma is a matter of months.

After having taken all these points into consideration, puncture with a sufficiently strong and wide cannula is justifiable.

In proliferating or hæmorrhagic peri-orchitis, a serous or morbid chocolate-coloured liquid or fresh blood is obtained ; from a tumour, however, nothing is forthcoming, or at most a few drops of blood, or a plug of tumour tissue may be found in the needle. The latter is useful for histological examination. If exploratory puncture yields nothing at one spot, whereas some mucous fluid is obtained at another spot, we should diagnose a *cystic adenoma.* If the fluid is of a light brown colour like *café-au-lait,* and contains epithelial cells, detritus and cholesterin crystals, the case is a *dermoid,* or at any rate an embryoma of similar constitution to the much more frequent ovarian dermoids, both of which may undergo cancerous degeneration.

The state of the testicle and epididymis is of no significance in cases of old-standing proliferating and hæmorrhagic peri-orchitis, because both these structures become atrophied and absorbed in the indurated cystic wall.

If the clinical picture of proliferating or hæmorrhagic peri-orchitis is combined with signs of acute local inflammation, with fever, and probably even with rigors, we should remember that these forms of peri-orchitis may easily become infected, and we should inquire whether there has been any therapeutic interference, such as puncture or injection of iodine.

(*b*) The diagnosis is very much easier when there is a solid tumour, definitely connected with the testicle and independent of the epididymis, or when both testicle and epididymis are fused together into one uneven tumour. If neuralgic pains are present in the spermatic cord there is no doubt about its malignancy; but it is not always possible to tell from the clinical signs whether it is **sarcoma** or **carcinoma,** a differentiation which is not always easy, even after histological examination.

A cystic adenoma or cystoma of the testicle is a very rare tumour, but we should bear it in mind if the swelling is irregularly roundish and nodular and contains both hard and soft areas, and if the above mentioned mucous fluid is obtained on exploratory puncture.

It is important to distinguish cancer and sarcoma from the swellings described by Wilms as *embryoid tumours*, which are derived from all the three layers of the embryo and grow in an erratic manner. The dermoids or embryomata just mentioned are innocent tumours, but these embryoid tumours behave clinically like cancer, and can only be distinguished microscopically. A tumour of an inguinal testicle may very probably be of this nature, at any rate, such has been my experience.

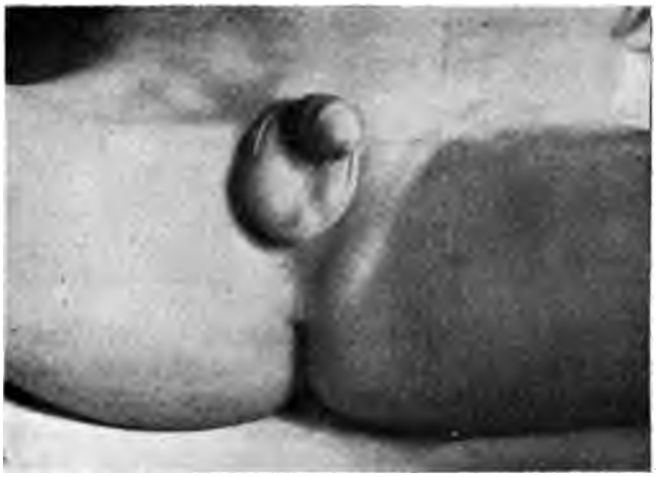


FIG. 251.—A displaced testicle in perinæum.

Tertiary syphilis of the testicle—either as a single gumma or a diffuse gummatous sclerosis—is the only disease which may lead to error in regard to moderately sized tumours limited to the testicle. If there are no metastases, and if we have no very good reason to definitely exclude tertiary syphilis, iodide of potassium should always be given. But if no result follows, operation must be undertaken, because cancer of the testicle soon gives rise to secondary deposits, and therefore the organ must be removed without hesitation.

The foregoing remarks have been made on the assumption that the testicle is in the scrotum. This, however, is not always the case. As we have already indicated, it may be retained anywhere along its route of descent—*retention in the abdomen* or in the *inguinal canal.* It may also have deviated in its course of descent, and be displaced, in which case it is generally found in the perineum (fig. 251). Any of the previously described pathological changes may occur in an abnormally placed testicle. Indeed, it is said that cancer is more liable to develop in a misplaced testicle than in one in a normal position.

A word with reference to **accidents.** How much attention should be paid to the assertions of insured patients that these various diseases have come on after an injury ? It is undoubted that even tumours may have a traumatic basis. Such a sequence is, however, very rare, and it is very necessary to ascertain in every case whether there was not some morbid change present before the accident. This also applies to tubercle. It is also a fact that the onset of gonorrhœal epididymitis is favoured by injury ; but this is no justification for claiming workmen's compensation, when the testicle has only been subject to the ordinary impact it may encounter during work, and its possessor has previously provided the necessary gonococci. This would be a defiance of the aims of accident insurance.

CHAPTER LIX.

FISTULÆ IN THE PERINEAL REGION.

The perineal region, the seat of various natural apertures, is also the gathering place of various fistulæ, some of which are congenital, while others only appear in later life. The principle of classification which we will adopt is based upon the site of origin of these fistulæ. The period of their appearance is not a satisfactory basis for classification, because even congenital fistulæ may not develop fully for many years. Neither can classification be based upon their position, because similar fistulæ may have very different situations. Sometimes microscopic examination is required to decide the nature of the discharge from the fistula.

We distinguish :—

(1) DERMOID FISTULÆ.

A fistulæ in the coccygeal area, discharging a small amount of secretion, which has been present for many years and which admits a probe into a short blind sac, is a dermoid fistula. The diagnosis is confirmed if on microscopic examination there be found not only pus, but also pavement epithelium and even hairs.

The patient who had the fistula depicted in fig. 252, extracted many hairs from it, with the aid of a mirror.

Apart from these embryonic invaginations, there is often found in this region a depression, the *foveola coccygea* (fig. 252) which corresponds to the point of attachment of the caudal ligament to the skin.

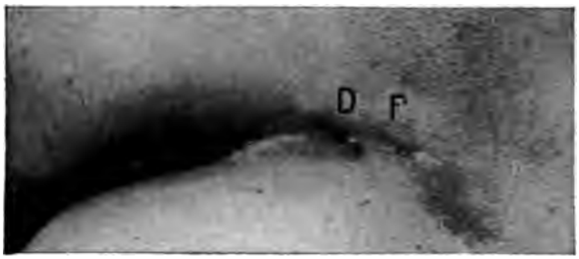


FIG. 252.—D = Dermoid fistula. F = Foveola coccygea.

(2) FISTULÆ IN CONNECTION WITH BONE.

These are almost always tubercular, and usually originate in the sacrum or ileo-sacral joint; more rarely in the coccyx or spinal column. The discharge from these fistulæ is always purulent, and the probe introduced reaches down to bone.

We can draw no conclusion as to the origin of these fistulæ from the histological demonstration of tubercle, because many simple rectal fistulæ are tubercular, and it is just from these that the differential diagnosis has to be made. Neither do negative results justify any definite conclusion, because the fistula may still be coming from bone, affected by osteo-myelitis.

Even if a fistula comes from bone, we are not justified in excluding the possibility of all connection with the rectum. The abscess may have opened secondarily into the rectum before appearing at the perinæum.

An unequivocal proof for its bony origin is only obtained if a skiagram shows the presence of a primary focus in the bone, or if this focus (vertebro-ileo-sacral tuberculosis) begins to manifest clinical symptoms.

(3) FISTULÆ OF THE RECTUM AND ANUS.

(*a*) *Congenital Fistulæ.*—These may be referred to three types, in accordance wiih the degree of occlusion and the sex of the patient:—

(1) The anal aperture is itself reduced to the size of a narrow fistula, which opens either in the anal cleft, the scrotum or penis.

(2) The anus is closed, and the ampulla is connected by a fistula with the vagina, or more frequently with the vestibulum.

(3) The anus is closed, and the ampulla opens into the urethra. The state of the anal fossa and the position at which the fæces escape permit an accurate diagnosis to be made forthwith.

(*b*) *Acquired Inflammatory Fistulæ.*—These are usually the final results of peri-rectal abscesses, which open externally near the anal aperture, and generally also break through somewhere in the rectum. They cannot be recognized, as a beginner might suppose, by the escape of fæces therefrom, for they are usually much too small for this. Besides, many of them are situated entirely below the sphincter, and others, despite their name, have no opening in the rectum. A fistula which has burrowed through, and opened above the sphincter, may, however, sometimes allow flatus to pass involuntarily. Before a fistula can be designated either rectal or anal, it ought to be shown that the original inflammatory process started in the rectum or anus. This is not possible, as a rule, and therefore our diagnosis is eventually made by exclusion; that is to say, a fistula in this region is either rectal or anal in the narrow sense, if we can find no other explanation for it.

The portal of entry for the infection is very varied. The infective process may have become engrafted on a fissure, a hæmorrhoid, an accidental wound, a simple eczema, or even on a urethritis. In other cases the infection probably attacks peri-rectal tubercular glands.

At other times the infection may enter through the folds of Morgagni (Chiari), or through Hermann's sinus, in which certain glands described by this observer open—this sinus itself opening into Morgagni's folds (Tavel). These fistulæ are invested with pavement epithelium.

In order to determine whither the fistula leads, the bowel must be emptied and the patient placed in the lithotomy position and a moderately thick probe is passed into the fistula with one hand while the index finger of the other is in the rectum for purposes of control. If we obtain no result the examination must be repeated after the introduction of a rectal speculum. If no connection is even then visible a sausage-shaped roll of gauze should be gently put into the rectum as high up as possible and the external opening of the fistula injected with a few cubic centimetres of a 1 per cent. solution of methylene blue. If there is any connection between the fistula and the bowel there will be a blue stain on the gauze when extracted, which will also show its position. If there is no connection we must see whether the probe reaches bone, and if not, must observe whether the injected solution appears in the urethra instead of in the rectum. If all these examinations yield negative results we must still regard the fistula as a rectal fistula, although it does not open into the rectum. But, on the contrary, we must not suppose that every fistula which opens into the rectum is a rectal fistula, as we have already indicated. Both fistulæ from bones and from the urinary tract may form secondary connections with the rectum.

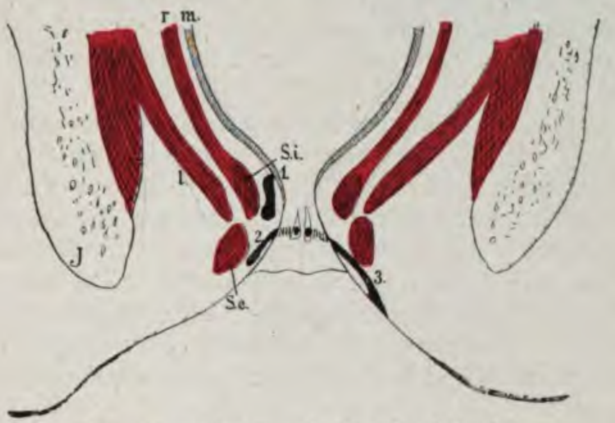


FIG. 253.—Diagrammatic view of anal fistulæ.

m = Mucous membrane of rectum.
r = Muscular layer of rectum.
Si = Internal sphincter.
Se = External sphincter.
l = Levator ani.
J = Ischium.
1 = Incomplete submucous fistula.
2 = Incomplete subcutaneous fistula.
3 = Complete subcutaneous fistula.

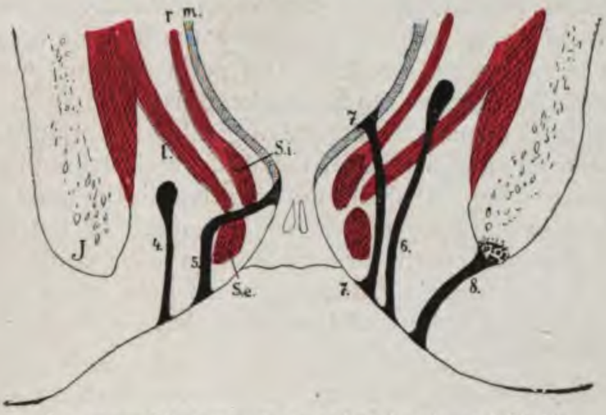


FIG. 254.—Diagrammatic view of anal and rectal fistulæ.

Anatomical details as in fig. 199.
4 = Incomplete ischio-rectal fistula.
5 = Complete ischio-rectal fistula.
6 = Incomplete pelvi-rectal fistula.
7 = Complete pelvi-rectal fistula.
8 = Fistula leading from tuber ischii.

A fistula which penetrates right through is called *complete*, one which ends blindly is called *incomplete*. If it runs between the external sphincter and the skin or the mucous membrane we speak of a subcutaneous or a submucous fistula respectively. If it lies outside the external sphincter and under the levator ani we call it *ischio-rectal.* If such a fistula is complete it usually opens just above the external sphincter, between it and the internal sphincter. If a fistula pierces through the levator ani it is called *pelvi-rectal.* If there is an internal opening to it, it will be found above the internal sphincter (fig. 254).

In regard to the treatment and prognosis we are confronted with the question whether the disease is tubercular, as it is in half the cases, or whether it is not. The very appearance of the fistula may convey some meaning to the careful observer and he will entertain a deep suspicion of tubercle if the skin is undermined and the granulations pale brown, just as he would if apical tuberculosis had been found beforehand, although the patient may have had no presentiment of it. Positive proof is afforded by histological examination of the granulations, and in doubtful cases by animal inoculation.

(4) URINARY FISTULÆ.

These are easily recognized and easily diagnosed, because a discharge of urine is an unmistakable symptom, and one which the patient realizes at a very early stage.

(*a*) We will begin with *congenital urinary fistulæ.* These obviously only occur in males and are caused by the urethra opening in the perinæum—hypospadia, scrotalis and perinealis—or in the rectum. This hypospadias merges into hermaphroditism.

(*b*) Acquired urinary fistulæ are of greater importance. We shall not refer to urogenital fistulæ of females, which belong to the department of gynæcology, but shall limit ourselves to the male sex.

The first question to decide is the source of the fistula. The mode and manner of the urinary flow may be conclusive on this point. If it is constantly dripping the fistula must run into the bladder, or some previous disease must have destroyed the action of the sphincter.

This form of fistula is rare. I have seen it in an old prostatic patient in whom a retroprostatic pouch had led to an extravasation of urine towards the perinæum, and therewith to the formation of a fistula.

On the other hand, if urine only exudes at the time that the patient micturates voluntarily, not, however, through the natural opening, but through more or less numerous fistulæ, it follows that the urethra must be leaking between the sphincter and some obstruction peripheral to it.

The spot at which the urine escapes is a matter of indifference. One finds sometimes, as a result of extensive urinary infiltration, fistulæ on the scrotum, penis, perinæum, lower abdomen, and even on the thigh, so that the patient eventually micurates as from a basin full of holes. If there is loss of sphincter action owing to inflammatory mischief the urine is constantly trickling from the fistulæ.

If the distinction between fistulæ from the bladder and from the urethra is once made, the etiology follows naturally. In cases of vesical fistula the obstruction must be at the neck of the bladder, and consists either of a tumour or prostatic hypertrophy. In cases of urethral fistula the obstruction must lie peripherally to the neck of the bladder, and consists of gonorrhœal or traumatic stricture, rarely of tubercle or cancer ; but in the Tropics, Bilharzia disease is frequent.

The fistulæ which develop after open injuries, and which concern the urethra rather than the bladder, follow no rule. The same applies to the fistulæ which develop in consequence of malignant growths of the urethra or anus.

CHAPTER LX.

GENERAL REMARKS ON THE SURGICAL DISEASES OF THE URINARY ORGANS.

THE cystoscope, catheterism of the ureters, the intravesical separation of the urine, and cystoscopy, may have improved the accuracy of diagnosis of urinary disease, but it has certainly robbed it of simplicity, and there is a risk that the practitioner will think that urinary disease, with the exception of nephritis, cannot be diagnosed without all these accessories. This would be, however, a grave error, and the exaggerated significance which was at first attributed to some of these methods is to blame. The general practitioner still sees urinary troubles in their earliest stages, and the responsibility rests upon him of recognizing when surgical assistance is required. Every practitioner has, or should have, a microscope at his disposal, and is conversant with the elementary methods of the chemical and bacteriological examination of the urine. These two aids, in addition to careful clinical observation, permit the diagnosis to be made in most cases sufficiently early for timely surgical treatment—if this is practicable.

We will not proceed to consider ready-made diagnoses like the headings of a text-book, but will take the symptoms which lead the patient to the medical attendant. These consist either of some disturbance in micturition or of some abnormal constitution of the urine. Then there come into consideration local symptoms in the area of the diseased organ.

I.—DISTURBANCES OF MICTURITION.

Micturition may be *painful* (dysuria) or *difficult* (retention), or, on the other hand, it may be *too free* (incontinence), or, finally, there may be a *persistent strangury* (tenesmus). *Involuntary* micturition, which is otherwise normal, has only rarely a surgical interest.

(A) PAINFUL MICTURITION.

Pain on micturition may originate either in the urethra or in the bladder and its vicinity. We distinguish the following possibilities :—

(1) If the urine scalds when it passes through the *urethra*, there is either some abnormality in its composition (concentrated or chemically changed), or the urethra is inflamed. The former condition may be caused by indulgence in certain beers, or by taking beer to which one is not accustomed ("biertripper"). In recent urethritis it will not, as a rule, be difficult to fix the blame on the gonococcus.

The observer must convince himself of the condition of the urethra by inspection, lest he treat a gouty urethritis for a gonorrhœa. The differential diagnosis is easy, because there is no discharge in the former, whereas in the latter it is always present in some form.

A pain which originates in the bladder is sometimes referred to the urethra, and the patient complains of a scalding in the glans penis, when, for instance, the bladder is irritated. Pain on micturition may also arise from some localized disease of the urethra, a stone, a foreign body introduced from without, or rarely from the early onset of carcinoma.

(2) Pain in the *neighbourhood of the bladder*, especially at the end of micturition, indicates disease in the bladder itself or in its vicinity.

(*a*) Stone and tuberculosis are the principal *diseases of the bladder* in this connection. Tumours do not cause pain until their later stages, unless cystitis supervenes. In tuberculosis, and more especially in stone, the pain occurs chiefly at the end of micturition, remains a long time after the completion of the act, and may radiate into the urethra.

In cases of stone there is the very significant circumstance that the pain and the accompanying strangury increase with bodily movements, such as riding on an uneven road.

(*b*) When the inflammatory process is in the *area adjacent to the* *bladder*, as in the case of perimetritis or of appendicitis, &c., wherein the bladder is directly involved in an abscess wall, the pain occurs at the beginning of micturition and remains more limited to the neighbourhood of the bladder; patients with appendicitis often retain their urine for many hours to avoid this pain. The same applies, although in a less degree, to the bladder pain which often occurs in cases of tubercular peritonitis.

(B) DIFFICULT MICTURITION.

This is caused either by some disturbance of the mechanism or by obstruction in the passage.

Whenever there is no urine passed, it is most important to show, by percussion or catheterism, that there is urine in the bladder, and that the failure of micturition is not due to suppression of urine. We have already discussed in connection with abdominal injuries the apparent anuria which occurs in laceration of the bladder.

(1) Disturbances of the Mechanism of Micturition.

Retention, due to disturbance of the mechanism, by an interruption in the reflex arc or by cerebral inhibition, is of medical rather than of surgical interest. The surgeon often sees this form of retention after operations, due to psychical inhibition.

It is not at all necessary that the operation should be on the genitals, urinary tract or their vicinity. Retention may also follow other operations such as excision of a goitre, or radical cure of hernia. Sometimes it is only the horizontal posture which disturbs the patient; in other cases physical and psychical reflexes of various kinds are responsible.

The retention observed in semi-comatose patients, especially in the course of meningitis, indicates functional disturbance of the reflex process. The voluntary retention in cases of painful micturition previously noted is quite of a different character. In cases of retention due to over-distension of the bladder, the cause is partially due to disturbance of the mechanism of micturition, but as the chief cause is some mechanical obstruction, we shall discuss this type of retention in the latter group.

Anatomical destruction of the nerve tracts is found in injuries of the spinal cord, and in compression thereof by tubercular caries or tumours.

(2) Obstruction of the Urethra.

The difficulty caused by mechanical obstruction of the urethra has much greater surgical importance. The following comprises a summary of the causes of such obstruction; foreign bodies and stone, inflammatory and traumatic strictures, compression of the urethra from without by a hæmatoma, tumour or inflammatory material, &c., and laceration of the urethra. Each one of these conditions possesses its own peculiarities, which, as a rule, render the diagnosis quite easy.

(*a*) A sudden onset, accompanied by pain and possibly by the passage of blood, strongly suggests a *foreign body in the urethra*. It may be a stone from the bladder, which has lodged in the urethra, a very probable contingency if there be a history of the passage of stones or of discomfort due to stones. But a foreign body may have been introduced from without, obstructing the urethra either by its size or by the inflammation which it provokes. History is usually silent in these cases, and nothing less than the ocular demonstration of the foreign body suffices to extort a confession. A metal catheter and a urethroscope are required for the diagnosis. The extent to which sexual perversion may go is shown by a case in our clinic, wherein the patient filled his urethra, as far as the sphincter, with plaster of paris.

Stones may remain in the urethra for a considerable time without leading to obstruction. These are stones lying in diverticula and their symptoms are of a chronic nature.

If the urethra is free, the cause of a sudden obstruction must be at the exit from the bladder, and then stone at the vesical neck is most probable. In certain positions such a stone may block up the neck of the bladder like a ball-valve. In most of these cases the patient will already have discovered that he can only micturate easily in a certain posture of his body. Often the micturition will be suddenly interrupted, or the previously powerful stream becomes suddenly feeble.

If obstruction has apparently come on suddenly at the neck of the bladder, and it remains absolutely unchanged as a complete obstruction for a day or more, we should think of **enlarged prostate** if the patient is an elderly man.

A careful inquiry into the history of these cases will elicit the fact that there have been symptoms of mild obstruction previously, but that the patient has not appreciated their importance. The sudden obstruction is also partially due to over-distension, and therefore to disturbance of the mechanism of micturition.

(*b*) *Subacute obstruction of the urethra* is the term applied to those cases wherein the process develops, without any warning, in the course of a few days. They are usually caused by rapidly growing swellings which press on the urethra from without—in males, by **abscesses of the prostate or vesiculæ seminalis**, or **peri-proctal**

suppuration: in females, by some **genital tumour** strangulated in the true pelvis and becoming rapidly larger through circulatory disturbances, by a pregnant retroflexed uterus, or by some **effusion** under high pressure.

(*c*) *Gradual obstruction of the urethra* presents quite a different clinical picture.

The patient complains that for weeks or months he has had to strain during micturition, and that the stream does not reach as far as formerly. Attacks of catarrh of the mucous membrane or of over-distension of the bladder may increase the symptoms spasmodically, but the obstruction may become complete quite suddenly, and its degree never varies, as it does in cases of obstruction by stone.

The causes of this gradual obstruction include stricture, new growth, stone in a urethral diverticulum, enlarged prostate, pelvic tumour, or a very chronic abscess. The age and history of the patient will suggest the selection from these causes, and local examination, which we shall describe later on, will allow us to make a more definite diagnosis.

(C) DEFICIENT CLOSURE OF THE BLADDER.

Inability to retain the urine, *incontinence*, may be due to many causes, some of which are concerned with medicine, others with surgery.

Disturbances of innervation are the most important of these causes. They may be of a purely psychical character and transitory in nature (fright or excitement). In other cases there may be organic paralysis of the sphincter, in which condition the incontinence is not primary, but merely the result of retention with overflow, from an over-filled bladder (paradoxical incontinence). The urine dribbles away, yet the bladder may reach as far as the umbilicus. Retention with overflow also occurs when mechanical obstruction has led to over-distension of the bladder, e.g., in enlarged prostate.

Incorrect diagnosis of these various disturbances is quite frequent. Pure incontinence as a result of sphincter paralysis is confused with overflow from a full bladder due to paralysis of the detrusor. The difference is, however, very obvious, because in the former case the bladder is empty, while in the latter it is full. The constant micturition which occurs when the capacity of the bladder is very small (as in tuberculosis or stone) is sometimes mistaken for genuine incontinence. Finally, pure nervous derangements may be regarded as the result of mechanical obstruction, and a patient with masked tabes diagnosed as a case of enlarged prostrate. These mistakes can only be avoided by a complete examination of the patient.

Ulcerative destruction of the sphincter by new growth or tubercle may also lead to constant flow of urine.

(D) VESICAL TENESMUS

must not be confused with incontinence. This term is applied to all cases wherein there is increased frequency of micturition, accompanied by abnormal sensation of irritation. The first thing which usually strikes the patient is that he is bound to get up once or more in the night, although he has not taken more than his average allowance of liquid. Then, he begins to be annoyed by frequency of micturition during the day, and, finally, strangury sets in, which prevents him from attending to his occupation.

The bladder is in a constant state of contraction owing to some persistent irritation. It is unable to fill up, and the urine escapes at short intervals, although the sphincter still retains the full power of closure. The main cause of this condition is cystitis, especially of tubercular origin, but it is sometimes due to a large stone in the bladder. In persons with extreme reflex irritability, such as is attributed to Rousseau, there is no relation between the physical state and the degree of vesical contraction. Even a small ulcer may suffice to produce the so-called "irritable bladder." The diagnosis is based on the frequency of micturition and the diminution of the capacity of the bladder, which can be demonstrated by the injection of fluid.

Whereas a normal bladder easily holds 200 to 250 c.c., an irritable bladder sometimes rebels against one-tenth of this quantity, and it is often quite impossible to inject the 80 to 100 c.c. which are required for cystoscopy, at any rate, without the aid of morphia or anæsthesia.

As the various conditions which lead to the anomalies of frequent micturition are very liable to confusion, they may be briefly summarized once again :—

Anuria.—Absence of urinary secretion, or its retention in the kidneys (the former in severe nephritis, the latter in stone in both kidneys); but the power of emptying the bladder is *not* lost.

Oliguria.—Very small amount of urine secreted, without regard to the frequency of micturition (e.g., in nephritis, ileus and diarrhœa).

Polyuria.—Increased secretion of urine, without reference to the frequency of micturition (e.g., in diabetes).

Pollakiuria.—Abnormal frequency of micturition, without reference to the quantity of urine. This may be the result of :—

(*a*) Abnormal filling of the bladder, with incomplete micturition (e.g., enlarged prostate).

(*b*) A condition of abnormal irritability (vesical tubercle, vesical stone).

(*c*) An abnormally large quantity of urine.

II.—ABNORMAL COMPOSITION OF THE URINE.

The substances which a patient notices as abnormal constituents of his urine are pus, blood, and inorganic deposits in the form of concretions.

(A) ADMIXTURE WITH PUS.

The naked eye should never be relied upon for the diagnosis of pus ; it is always necessary to employ chemical examination and the microscope. This, however, does not mean that the naked eye cannot discover a good deal in a cloudy urine.

The following type of case is not infrequent : A patient consults us for a condition which has been diagnosed, either by himself or by others, as cystitis. He complains of tenesmus, and in evidence of his disease he puts on the table a bottle containing whitish cloudy urine. While describing his symptoms in full detail, a sediment forms in the bottle, which the experienced observer sees at once is not pus, but *carbonates* and *phosphates*. A few drops of acid dissolve this precipitate. A microscopic examination of the deposit will reveal amorphous calcium salts, and probably also the beautiful crystals of di-calcium phosphate ; sometimes also calcium oxalate. A few general directions for the *regimen* of the body and mind suffice to cure the patient of his "cystitis."

If the urine is slightly cloudy, but forms no deposit even after long standing, nor clears up on the addition of acid it is most probable that the specimen is not fresh, but has become a culture medium for bacteria. A glance with the microscope shows us swarms of bacteria but no pus cells. If the patient assures us that the somewhat cloudy and offensive urine has been passed quite recently, the probability is that the bacterial culture has developed in the urinary passages, and another examination with a catheter specimen would prove this. Pus cells are entirely absent, neither are there any clinical signs of inflammatory disease of the urinary passages. The case is one of "*bacilluria*," generally due to the *Bacillus coli* but sometimes to the typhoid bacillus.

If there really be *pus* in the sediment, the urine must be examined very carefully with the naked eye. If gonorrhœal threads or small flakes of pus float about in it, the urethra of the patient must be examined, and, according to circumstances, the urine must again be investigated after washing out the urethra, or the three-flask test must be undertaken. If the pus only comes from the anterior urethra, or from the posterior urethra as well, leaving the bladder free, the case is gonorrhœa. If both the urethra and the bladder discharge pus, we shall rarely err if we diagnose cystitis as a complication of gonorrhœa. But if all the pus comes from the bladder, we must inquire whether there is always a sediment in the urine, or whether the cloudiness in the specimen under examination is a first appearance, or whether such cloudy specimens have been passed on previous occasions. If the urine is uniformly purulent, or at any rate if the pus contents are not subject to sudden changes, the lesion may be in the bladder, although the pus may come from the pelvis of the kidneys.

A single evacuation of very purulent urine, or of pure pus, indicates, either that a pyonephrosis has suddenly emptied into the bladder, or that a perivesical abscess has broken into it. Such an abscess may originate in the appendix, female sexual organs, prostate, or pelvic bones. This event will always have been preceded by symptoms which permit of the establishment of a diagnosis. The appearance of pus in the urine is only an incident in the course of the underlying disease. The site of the perforation can be seen with the cystoscope.

We now proceed to the *chemical examination of the urine.* The chief information which this affords, apart from the demonstration of carbonates and phosphates previously mentioned, concerns the reaction of the urine. Diminution of acidity, or even the presence of an amphoteric reaction, has no serious significance, as long as the urine is odourless, and if any cloudiness which may be present disappears on the addition of acid. On the other hand, a diminution of acidity in pus-containing urine, and the presence of an alkaline reaction, indicates secondary infection of the urinary passages by organisms like the staphylococci and *Proteus vulgaris,* which decompose urea. The latter causes, in addition to an alkaline reaction, ammoniacal fermentation, which betrays itself at once by the smell. If pus-containing urine is acid, but does not smell offensively, the condition is usually tubercular or one of streptococcal infection. If it is acid and offensive in smell, the *Bacillus coli,* and probably other inflammatory organisms, are responsible.

It goes without saying that the chemical examination of the urine must always include tests for albumin and sugar, and for biliary and blood pigment when necessary.

The most important part of the *microscopic investigation of the urine* concerns the various *forms of cells.* If polynuclear leucocytes preponderate, the morbid process is an acute one; if mononuclears preponderate we should rather think of tuberculosis. Bladder epithelium indicates the presence of ulcers, especially if it is found in shreds, and if specimens of the deeper layers occur (caudate epithelium). Red blood corpuscles point to the same conclusion. Whenever pus cells are present, cylindrical cells should always be sought for.

It is then necessary to note the presence of various kinds of *micro-organisms.* The demonstration of tubercle bacilli posseses a special significance, and to prevent their confusion with smegma bacilli, a catheter specimen must be obtained, a precaution requisite in all bacteriological investigations of urine. If nothing is found, this is naturally no evidence against tuberculosis. But we must not hastily conclude that the suppuration has been adequately explained by the discovery of the *Bacillus coli*, staphylococci or streptococci. These organisms may certainly exist independently, but they often accompany the tubercle bacillus, even in uncatheterized cases, and may indeed completely overshadow it in the urine. We may even go a step further and say that every case, wherein these organisms persistently occur in the urine, is very suspicious of tubercle. The suspicion is even greater, if only pus cells are found and no micro-organisms at all. Such cases are almost always tubercular. This diagnosis is obviously arrived at by a process of exclusion, but it can be confirmed by the inoculation of guinea-pigs—a procedure which should never be neglected if tubercle bacilli are not found in the direct examination of purulent urine, in cases wherein there is no other cause for the suppuration. If this experiment, conducted with an adequate amount of sediment, yields no result, we are then justified in assuming that the case is one of pyelitis due solely to the "ordinary" pus organism.

(B) ADMIXTURE WITH BLOOD.

The presence of blood in the urine is always a serious matter, whether there are only a few red cells detected by the microscope, or whether there has been profuse hæmorrhage.

(1) If the blood is quite red and also flows independently of micturition, it comes from the urethra and must be ascribed to injury, possibly by a foreign body.

Smaller periodical hæmorrhages independently of micturition indicate some ulcerative process in the urethra (cancer, or stone in a diverticulum). The appearance of *blood-stained semen* should also be mentioned. Cases occur wherein there is a discharge of blood-stained semen, quite apart from any sexual activity, and even after the period of sexual life has been left behind, although no objective demonstrable disease of the sexual organs be present.

(2) If the blood is mixed with the urine and appears only with micturition, it must come from the *bladder*, *ureter*, or *kidneys*. Attempts have been made to decide whether the blood comes from the kidneys or the bladder, on the basis of the more or less pronounced change in the colour of the blood. These are, however, unreliable, because everything depends on the rapidity of the hæmorrhage, the amount of blood, and the duration of the contact between the urine and the blood. If the hæmorrhage in the bladder is slight, and the blood remains within it for any considerable time, it under-

goes the same changes as occur in hæmorrhage in the pelvis of the kidney. Much more reliable conclusions may be derived from the associated symptoms. If the hæmorrhage is attended by *vesical colic*—due to the expulsion of coagula—and by *no other* symptom, the blood most probably comes from the bladder. If *renal colic* is present, its origin is in the kidneys. If there be no pain, nor any indications of tumour in the bladder or kidney, we may be able to obtain some information by examining the urine after massage of the kidneys. The last stage of the examination consists of cystoscopy.

(3) If there be pus in the bloody urine, or in the urine of the intervals wherein it is free from blood, we should think especially of tubercle, or of some vesical or renal disease with secondary infection, however slight the traces of pus may be.

(4) Microscopic traces of blood, constantly found in the sediment or the centrifugalized portion, indicate either stone or tuberculosis.

(5) Intermittent hæmorrhage and the presence of cylindrical cells and albumin in the intervals when the urine is free from blood, are signs of chronic hæmorrhagic nephritis.

Nephritis may, however, occur without albumin and without cylindrical cells, as shown by Rovsing. But we should only conjecture such a diagnosis in cases wherein the blood comes from both sides and cannot otherwise be explained.

It is also necessary to add, for the sake of completeness, that renal hæmorrhage may occur in hæmophilia and in transitory hæmorrhagic diatheses, as in purpura. Whether, apart from the above-mentioned conditions, bleeding may occur from healthy kidneys—idiopathic renal hæmorrhage—as is often assumed, must be left an open question. Such a diagnosis can only be made with certainty *post-mortem*, because anatomical changes cannot be excluded unless histological examination of both kidneys has been undertaken.

(C) ADMIXTURE WITH INORGANIC DEPOSITS OR CONCRETIONS. (URINARY GRAVEL.)

Inorganic sediment is the third form of urinary admixture which worries the patient and impels him to seek advice. This sediment varies from a flocculent deposit of microscopic particles and crystals, to the size of a pea, e.g., to a calibre which can just pass through the urethra. We have already referred to sediment composed of carbonates, phosphates and oxalates. In the case of "gravel" or larger concretions the diagnosis of calculous disease is obvious. But such a diagnosis should not satisfy us. It is much more important to decide whether we are dealing with an "aseptic" or with an "infected" case. In the former, the urine is free from pus and the concretions consist of calcium oxalate, uric acid, urates and occasionally of cystin.

In the latter, the urine is purulent and the concretions consist, exclusively or largely, of ammonium-magnesium phosphate and basic calcium phosphate.

Combustible	Murexide test	With ammonia—purple red With caustic potash—purple violet	Uric Acid Urates
	Murexide test	With ammonia—yellow With caustic potash—orange	Xanthin
	The powder burns with a slightly luminous blue flame, and smells like burning sulphur and oil of asafœtida		Cystin
Incombustible	The powder effervesces with hydrochloric acid		Calcium Carbonate
	The powder does not effervesce with hydrochloric acid	but does so after heating to redness	Calcium Oxalate
		and does not do so, even after heating to redness	Earthy Phosphate

The diagnosis can often be made from the microscopical examination, because the urine frequently contains, in addition to the peculiar granules of gravel, some crystals of the corresponding salts, illustrations of which are given in any text-book of clinical methods. If these crystals are, however, absent, the diagnosis can be made by the experienced observer without any difficulty, from the above scheme suggested by Ultzmann.

The *origin* of these concretions cannot be determined forthwith, at any rate not of all of them. We may, however, assume that clinically aseptic concretions originate in the renal pelvis, while secondary stones may form either in the renal pelvis or the bladder. Gravel might come from either source, but small facetted burnished stones usually originate in the renal pelvis.

III.—LOCAL SYMPTOMS.

An accurate diagnosis can only be made after the direct examination of the organs concerned. We shall later on discuss the various groups of disease in detail, but for the present, will limit ourselves to a few remarks on the *method of examination*, and the so-called *functional diagnosis of renal disease.*

The patient must first pass a portion of his urine—if he can—and we put this aside for a careful examination, particularly to ascertain whether there is any admixture with blood. The character of the stream and the naked-eye appearance of the urine will already have furnished important information (gonorrhœal threads, pus, gravel). We then feel the urethra, lest we miss some foreign body, tumour, or scar tissue which may be felt from the outside, and afterwards we palpate over the bladder and kidneys. A wide Nélaton catheter is then introduced, having previously satisfied ourselves as to the condition of the instrument.

It is very unfortunate if the practitioner has to extract a broken piece of catheter himself, or obtain the services of some one else to do it for him.

The following are the usual possibilities :—

(1) If the catheter enters the bladder easily, although the patient cannot himself micturate, the neck of the bladder must be obstructed by a stone, foreign body, or tumour; or it may be a case of enlarged prostate, or compression of the urethra from without—assuming, of course, that a nervous derangement is not in question. In cases of enlarged prostate or external compression of the urethra, the catheter experiences some little difficulty when it reaches the pars prostatica. We then take a medium-sized metal catheter of the ordinary curve, and introduce it very carefully. If we reach the bladder and then impinge on a hard, rough substance, there can be no doubt about the diagnosis of stone or foreign body. If nothing is felt, we must notice whether it is necessary to depress the eye of the catheter very much before the urine flows. In this event the pars prostatica is lengthened, which means that an enlarged prostate is probably present. Sometimes it is necessary to elevate the patient's pelvis in order to depress the catheter sufficiently. The diagnosis obtains further confirmation if we cannot empty the bladder with the ordinary shaped catheter, but succeed in doing so with a semi-circularly curved tin catheter, or with an elastic catheter with Mercier's curve.

Having succeeded in introducing into the bladder a medium-sized or wide catheter, either with or without this special manœuvre, the question arises as to whether we are dealing with a simple prostatic hypertrophy, a prostatic tumour, or with some pathological structure in the vicinity, which is pressing on the urethra. The latter usually signifies a swelling, which is more often malignant than innocent, and includes tumours of the pelvic bones and connective tissue, such as sarcomata, chondromata, osteomata, and cysts, mainly dermoids. The distinction between these conditions and enlarged prostate is made by rectal and combined recto-abdominal examination.

With the finger introduced into the rectum, we first feel the anterior wall of the ampulla, and follow the outlines of the prostate with the finger tip. It is impossible to learn from books what this feels like; it must be studied on the living subject. If the mucous membrane is soft and œdematous, with the prostate enlarged and rather elastic, feeling like a pillow, and at the same time tender to pressure, the case is one of acute prostatitis or of prostatic abscess. If the swelling is higher up, the case is one of inflamed vesiculæ seminales.

If the prostate is enlarged, but not painful on pressure, the case is one of enlarged prostate, cancer, or sarcoma. If there is nothing special found in the prostate, the hypertrophy may be in the direction of the bladder with or without a middle lobe, or the prostate may be in a state of diffuse sclerosis, or there may even be a contracting cancer present, which will be referred to in more detail later on.

Otherwise there must be present one of the previously mentioned tumours, pressing on the neck of the bladder from without.

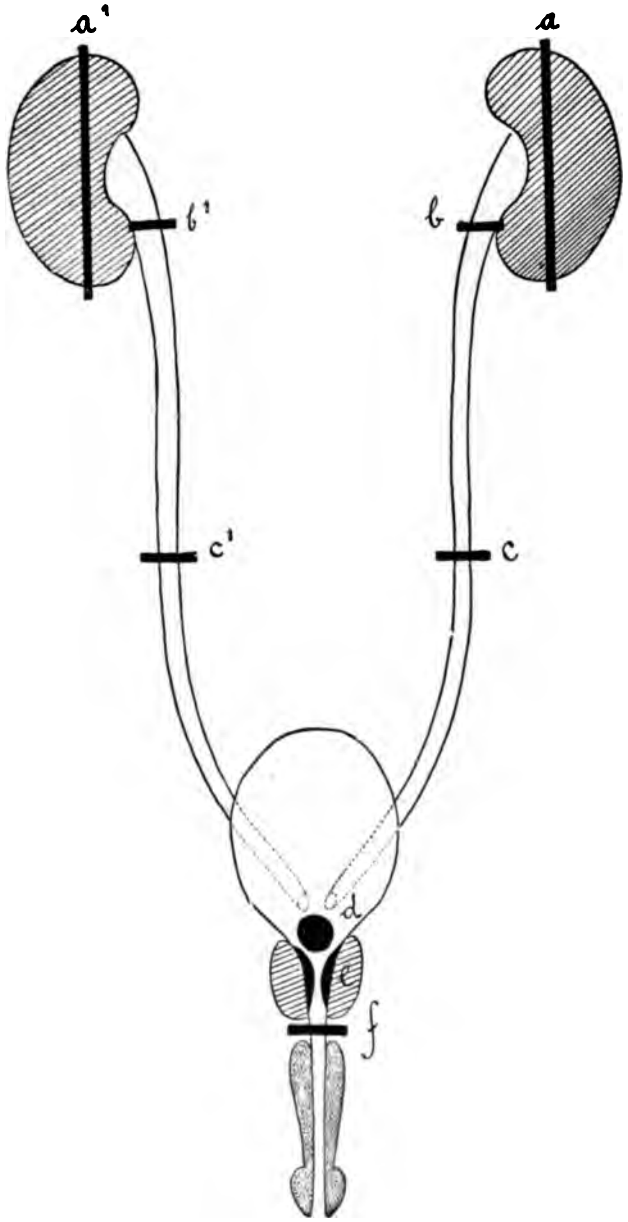
In the female sex, in addition to the pressure of the fœtal head during labour, it is necessary to mention uterine tumours incarcerated in the true pelvis, as well as retroflexion of the gravid uterus. Everyone of experience knows those tumours, reaching as high as umbilicus, which beginners look upon as ovarian cysts, but which disappear as soon as a catheter is passed. It is most important to make an accurate estimate of the state of affairs, as a very prolonged over-distension may result in complete sloughing of the mucous membrane of the bladder. A similar result may occur from rapidly growing tumours, strangulated within the true pelvis, and also from purely nervous causes.

(2) If a medium-sized catheter does not enter the bladder, but a narrow one does, a stricture is present. If it is not very definite, it is useful to employ Guyon's olivary bougie, because we are better able to appreciate the obstruction with this instrument than with a cylindrical or a cylindro-conical catheter. But if even the smallest catheter will not enter, we must try a series of elastic bougies of the calibre of catgut, putting in one after the other. A path will sometimes be found in this manner. The cause of the stricture is either gonorrhœa or new growth. If the age and history of the patient, and slight hæmorrhage from the stricture suggest cancer of the urethra, it may be possible to establish this diagnosis by palpating the urethra and the use of the urethroscope.

(3) If no instrument at all passes, the case is a severe example of one of the two above-mentioned classes. If the age, sex, and external circumstances do not give a clue, and if the over-filled bladder prevents a satisfactory examination, we must puncture the bladder as a matter of urgency, and then provide an exit as rapidly as possible, either above or below, in accordance with the physical condition found.

(4) If the bladder is easily accessible, and it is necessary to decide the nature of the vesical disease, or the source of blood or pus, we must employ the aid of the **cystoscope.**

This instrument shows us whether the mucous membrane is healthy, inflamed, covered with fibrin, or ulcerated. It also shows



(*a*) Unilateral inhibition of the renal function (trauma, infarct). Local symptoms. No marked disturbance of urinary excretion (function taken on by other kidney).

($a + a^1$) Bilateral inhibition of the renal functions (nephritis). Anuria. Bladder empty. Death from uræmia.

(*b*) Unilateral obstruction of renal pelvis (kinking owing to abnormal insertion, or floating kidney, blocking by stone). Unilateral renal colic. Hydronephrosis. No uræmia.

($b + b$) Bilateral obstruction of renal pelves (most frequently stone). Bilateral renal colic, anuria, uræmia.

($b + a^1$) Obstruction by stone, with reflex anuria. Same symptoms, but colic only unilateral.

(*c*) Blocking of one ureter. (Stone, pressure by tumours) Same symptoms as in *b*.

$c + c^1 = b + b^1.$
$c + a^1 = a + b^1.$

(*d*) Obstruction of neck of bladder by stone. Retention of urine. Bladder distended. Flow of urine variable. Catheterism easy.

(*e*) Neck of bladder obstructed by tumour. (Enlarged prostate, cancer, sarcoma.) Retention of urine, partial (residual urine) or complete. Large catheter usually passes easily.

(*f*) Obstruction by urethral stricture. (Trauma, gonorrhœa). Retention of urine. Only a small catheter can be passed, but this sometimes fails.

FIG. 255.—Diagram of the various surgical derangements of micturition.

us the position of ulcers which may be present, and if they are arranged around one ureteral orifice, it indicates to us which kidney is diseased. It also shows us the shape of the ureteral orifices, it tells us whether the wall of the ureter is thickened, and enables us to decide the character of the urine, which trickles at intervals from the ureteral orifice, in accordance with its transparency or cloudiness. By its means we are also able to see stones, foreign bodies, tumours, and diverticula. But it is indispensable that the bladder should be able to hold 80 c.c. of water, and that there should not be any severe hæmorrhage.

The technique of the examination is not described here, because the general practitioner cannot be expected to do more than have the examination carried out early enough for the successful issue of any surgical operation which may be necessary.

If, finally, we have to determine the functional capacity of the whole renal apparatus, or of each individual kidney, this is a matter of the so-called **functional diagnosis of renal activity.** This does not come within the province of the general practitioner, because it requires more technical practice, experience, and time than he has at his disposal. He will, however, desire to know how it is carried out, and what results it yields.

The functional activity of the total renal parenchyma is first estimated, and then that of each individual kidney.

The first task demands the *estimation of the freezing point of the blood*, according to Koranyi. The lower this is, the more "urinary" substances there are in the blood, and the functional activity of the total renal apparatus is correspondingly unsatisfactory. The normal freezing point is 0·59, but the lower limit of 0·56 is allowed. There are many sources of error in the method, but in some cases it is useful. Important information may be derived from the estimation of the amount of urea passed in a day on a definite diet, and by testing the permeability of the kidneys to water and to sodium chloride.

The solution of the second problem demands the separation of the products of the two kidneys, by means of an intra-vesical partition or by catheterizing the ureters. The *activity of each kidney* is then tested by determining the freezing point of the urine, by estimating the nitrogen, or by examining the reaction of the kidneys towards certain subcutaneous injections. Thus the time which it takes for a gluteal injection of indigo carmine (4 c.c. of a 4 per cent. sterile solution) to appear in the urine is estimated (normal time ten to fifteen minutes). Or a subcutaneous injection of ·005 grm. of phloridzin is given and the amount of sugar excreted by one kidney in a definite time is measured; or one notes accurately when the excretion begins. None of these methods as hitherto employed are quite free from objection, but the sum total of the conclusions which they yield may be of distinctive significance from the point of view of diagnosis and treatment. The determination of Ambard's constant, perhaps yields more mathematically accurate information about the total functional capacity of the kidneys. Ambard assumes that there is definite ratio between the body weight, the urea content of the urine, and the urea content of the blood, which he has expressed by an empirical formula, the reliability of which requires confirmation. But the method is not simple enough to be employed as an ordinary means of surgical diagnosis.

CHAPTER LXI.

INFLAMMATION IN THE NEIGHBOURHOOD OF THE KIDNEY.

It is necessary to begin with a word about nomenclature. An attempt has been made to distinguish between paranephritis and perinephritis, which were previously considered synonymous. It has been suggested to limit the term *perinephritis* to inflammation of the connective tissue capsule of the kidney, and *paranephritis* to suppuration within the fatty covering. Further, Israel has designated the inflammation which occurs exclusively between the kidney and retro-renal fascia as *epinephritis.* However logical these distinctions may be, they cannot be applied in actual practice, because these three different anatomico-pathological conditions do not present separate clinical pictures. Perinephritis, defined as above, is never a disease by itself, but is always an accompanying symptom, and has neither diagnostic nor clinical significance. It is almost impossible, clinically, to suspect any distinction between paranephritis and epinephritis. Such a distinction is only possible at the operation or the autopsy, and it really possesses no practical significance. We therefore will adhere to the term *perinephritis* as implying all inflammation between the kidneys, peritoneum and lumbar muscles—a uniformity of nomenclature which should obtain international sanction.

There are three stages of perinephritis, each of which gives rise to a special train of diagnostic considerations.

(1) The patient becomes ill with high fever and obscure symptoms, which he attributes to some malady in the loin, because that region is painful. If the vertebral spine is somewhat rigid and the lumbar muscles are contracted on one side, or if they contract on being palpated, we are in all probability confronted with a case of perinephritis.

In this stage the most frequent error arises from confusion with *pleurisy.* In the latter, however, the pains radiate towards the shoulder, whereas in perinephritis they radiate towards the half of the abdomen on the affected side, towards the external genitals and even as far as the thigh.

I have had a case wherein definite lumbar pain which radiated downward caused me to expose the kidney, whereas the real trouble was a commencing empyema which, however, afforded no clear local symptoms. The difficulty in diagnosis would be much greater if a purulent pleurisy supervened on an early perinephritis. The result obtained by an exploratory puncture is attended by the same difficulty of interpretation as occurs in cases of subphrenic abscess.

An abscess of the perirenal connective tissue is topographically sub-phrenic. It is however distinguished from a genuine sub-phrenic abscess (an encapsuled sub-phrenic peritonitis) by the absence of any intra-abdominal cause, and by the definite reaction of the lumbar muscles.

(2) In the second stage the diagnosis is much easier, because, in addition to the above symptoms, a resistance can be felt in the lumbar region. If this resistance is sharply defined, and round in contour, the inflammation is usually in the kidney itself. If it is diffuse and indefinite, the perirenal tissue is involved, although, of course, the kidney itself may also be affected. If we are doubtful about the definition of the swelling, because it appears to be too sharply defined for a phlegmon, and insufficiently defined for a kidney, we must note whether the swelling moves on respiration. A kidney always moves downwards on deep respiration, even if it is morbidly enlarged, provided there are no perirenal changes. A perirenal abscess remains immovable.

Sharply defined abscesses are otherwise generally of a tubercular nature, and are recognized by the slight pain which they cause on pressure and by the very moderate effect which they have on the temperature of the body.

(3) In the third stage, the abscess may open into the lumbar region, causing a subcutaneous phlegmon, or it may travel into the pelvic fossa, provoking flexion of the thigh, or, finally, it may reach the pleural cavity, and burst into a bronchus. In all these conditions, the diagnosis of abscess can hardly be mistaken, but its original source in such advanced cases can only be ascertained from the history.

We should not, in any case, be content with the diagnosis of *psoas abscess*, which used to be a very favourite one. Psoas abscess may be a tubercular burrowing abscess, an osteo-myelitic suppuration, inflammation arising from the kidney or intestine invading the muscle, or, finally, a phlegmon which has originated in the broad ligament; a psoas abscess never constitutes a disease of itself.

So far, our diagnosis has been directed to suppuration in the perirenal fatty tissue. We look for its origin particularly in the kidney, pelvis, or spinal column. But its source may also be in one of the intraperitoneal viscera—appendix, liver, gall-bladder or large intestine.

If the urine contain pus, we must refer the perinephritis to renal tuberculosis, nephrolithiasis or some other kidney disease attended by suppuration. If the history does not point to any previous kidney trouble, we should think of some acute metastatic renal abscess, and search for a primary source of infection, e.g., a furuncle, sore throat or eczema. In other cases the primary disease is some infectious disorder, such as typhoid fever, small-pox, &c. If nothing abnormal is found in the urine, this is not to be regarded as conclusive against a renal origin. If, despite the absence of any abnormality in the urine, there is a history of some old-standing renal disease, we must assume that the ureter on the affected side is blocked up.

If there be no evidence whatsoever pointing to the kidneys, we must examine the adjacent bony parts, not only when the abscess is chronic and bears tubercular characteristics, but also when the abscess has developed acutely, because this may be due to osteo-myelitis of the pelvis. Should nothing be elicited here, we must next think of the appendix, which, not infrequently, lies in a lumbar or even a prerenal position. In such cases we cannot attain to anything more than a probable diagnosis, unless the appendix had originally been intraperitoneal and given rise to typical attacks of appendicitis.

There is no difficulty in diagnosis in the cases wherein a phlegmonous parametritis, following an abortion or confinement, has extended as far as the lumbar region. Liver and gall-bladder abscesses rarely encroach upon the perirenal tissue, but such secondary abscesses are recognizable by their history and the localization of the antecedent inflammatory symptoms.

If no other cause whatsoever can be discovered, we may assume that the case is a primary perinephritis, i.e., an infection of perirenal tissue by micro-organisms of unknown origin, and without any demonstrable involvement of the renal tissue. These abscesses usually arise through the coalescence of small abscesses of the renal cortex, which cause no symptoms in themselves, and do not alter the character of the urine.

CHAPTER LXII.

MOVABLE KIDNEY.

At one time movable kidneys were very fashionable, and to undergo treatment for them was regarded as an evidence of good tone; but this is now a thing of the past. We are now better able to realize the significance of this condition than we were fifteen years ago, and although movable kidneys have been unjustifiably condemned for all kinds of ills, nevertheless they do raise important problems of diagnosis, which should be considered together.

Firstly, as to the *evidence of their existence.*

The term should only be applied to kidneys which have *acquired increased mobility,* and not to those which are the subject of congenital displacement—a matter already discussed. This acquired mobility may, however, often be due to congenital causes. Thus I have seen a movable kidney in a young girl, aged 11, who was otherwise in perfect health—an abnormality which was most probably the result of some congenital predisposition.

The following method should be adopted to demonstrate a movable kidney. The patient—usually a female—must lie flat and as relaxed as possible. The lumbar muscles must be supported with one hand, but they must not be allowed to become tense; the other hand is gently pressed under the costal margin against the spinal column, but care must be taken not to make the muscles contract. The patient is then told to breathe deeply with the diaphragm. In this way it is usually possible to detect the descent of the kidney. In some cases, however, the kidney is first felt at the moment it slips upwards into its bed. The examination is immediately successful if the patient is thin and is able to breathe according to instructions, but if she is fat and cannot carry out the abdominal type of breathing as requested, a little practice is necessary. If examination with the patient on her back yields no result, she must be turned on to her side—on to the left side for the right kidney—or she must be examined in the erect posture.

What is the *normal* degree of mobility?

This varies in the two sexes. In a male, it should hardly be possible to feel the lower pole, even of the right kidney; but in a female there is nothing abnormal in being able to feel the lower third. In slender women it may be possible to feel even half of the kidney, without the condition being pathological, whereas in a man this should certainly be regarded as an early stage of movable kidney. If the upper pole of the kidney can easily be felt, it is obviously abnormal.

We have been assuming that the structure felt at the side of the abdomen is really the kidney; but this assumption is not always correct. On the left side, we may be deceived by an intestinal tumour, but only if we fail to observe that the structure does not move with respiration. On the right side, error may arise not only from an intestinal tumour, but especially from a constricted lobe of the liver and from a tensely filled gall-bladder. We have, however, discussed these possibilities in connection with the surgery of the liver and biliary passages, to which section the reader is referred.

Exceptionally, it may be quite impossible to arrive at a decision. If the diagnosis is important from the point of view of treatment, some assistance may be derived from a skiagraphic examination, after introducing a ureteral catheter, opaque to X-rays, or a 5 per cent. collargol solution as far as the renal pelvis.

As clinical and experimental experience have shown that this latter procedure may easily damage the kidney, it needs the greatest care in its employment. The introduction of the solution must be conducted so as to avoid over-filling or too high a pressure, and some of the solution must be run off as soon as any signs of renal colic appear.

If the structure felt is really the kidney, we must next inquire whether it is responsible for the pains of which the patient complains, bearing in mind that most movable kidneys never cause any symptoms at all, even when the degree of mobility is great. On the other hand, we should remember that a movable kidney is not usually an isolated phenomenon, but is part of a general visceroptosis which is, primarily or secondarily, associated with a neurotic state which depresses the patient, both physically and mentally. Stiller calls this condition "constitutional asthenia," although, of course, this term does not explain it. The psychical and organic reflexes are abnormally irritable in this condition, and the slightest discomfort—sometimes even physiological processes—is felt as a severe pain, or, at any rate, complaint is made. But, nevertheless, a movable kidney may itself give rise to pain by attacks of so-called strangulation—a term which is meaningless and ought to be abandoned. These attacks are really due to intermittent hydronephrosis, which will be described in the next chapter, and which are produced by the sharp kinking or twisting of the ureter, as a result of the displacement of the kidney. But, apparently, paroxysms of pain may also be caused by kinking or twisting of the renal nerves, also a result of the abnormal mobility of the kidney. It is, therefore, better to speak of torsion, rather than of strangulation. The polyuria which usually follows these attacks, does not depend upon the discharge of urine which has been dammed back, but upon some reflex process, as is also the case in many instances of intermittent hydronephrosis. As abnormal mobility of the kidney may produce these severe paroxysms, it is only natural to suppose that milder pains may also be due to the same cause.

But this assumption should only be made under certain conditions, a main one being that the pain is limited to the affected side, or, at any rate, is strictly distinguishable from other pains of which the patient may complain. The pain should radiate towards the inguinal region, scrotum and thigh, in contrast to the pain of gall-bladder disease, which radiates towards the right shoulder. The principal point, however, is that the pain is increased by any movement which displaces the kidney considerably downwards (over-flexing the trunk backwards, raising the arms on high) and is relieved by the horizontal posture. The pain is often diminished by wearing an effectual binder on the lower abdomen (especially Glenard's abdomen binder) and also by the support of the gravid uterus.

If the patient describes attacks which appear to be due to torsion, we must wait until another one comes on, and examine the patient during its continuance, in order to determine whether the kidney is tender and swollen. We may then find that the pain has nothing to do with the kidney, but that it indicates an attack of mucous colitis. The same care and repeated examinations of the bowel and stools are especially necessary when the pains vary between the right and left side, although the movable kidney is unilateral. Such pains are nearly always of intestinal origin, and are usually accompanied by alternating diarrhœa and constipation, and by the passage of some mucus. To attempt to stitch up the sunken organ in such individuals, would usually mean to operate on the kidneys, stomach, colon, liver and uterus. The result would probably be a failure, because the pains due to excessive mobility would merely be replaced by pains due to adhesions. It is this kind of experience which has damped the enthusiasm of those who were staunch advocates of stitching-up displaced viscera, and it is now recognized that a movable kidney does not necessarily require stitching, because its anatomical disposition is not the only matter to be taken into account. These patients principally require a rational diet and a natural mode of life to invigorate their tissues and improve their nervous system. We may, indeed, prescribe these remedies, but we cannot secure them for the patients, because some are too low in the social sphere, while others are too high to live a rational healthy life. There is a further type among these patients, which is absolutely uninfluenced by any kind of therapeutic measure.

CHAPTER LXIII.

HYDRONEPHROSIS AND ITS CONSEQUENCES.

THE retention of urine in the pelvis of the kidney produces a number of clinical pictures, varying with the conditions under which it has arisen, and each one gives rise to its own diagnostic problems. We may distinguish :—

(1) **Closed Hydronephrosis.**—This appears as a tense swelling, situated in the hypochondrium, and its differential diagnosis is discussed in the chapter on Abdominal Tumours. It is only necessary to add that, in rare instances, cystic swellings which do not depend upon retention in the renal pelvis, make their appearance in the kidney region. These are the congenital cystic kidneys (see also under Renal Tumours), which are distinguishable from hydronephrosis by their nodular surface, and which are often associated with a cystic liver. One should also think of hydatid cyst in districts where this is endemic.

As has already been observed in connection with hydatid of the liver, unexplained attacks of urticaria may suggest this diagnosis.

If the sac of a hydronephrosis becomes infected through the blood-stream, it develops into a closed abscess with all the symptoms of pus retention. Unless an exit is made for the pus, the perirenal tissue may become infected, and, finally, also the pleura. The following case is typical :—

A middle-aged female was suffering from a movable tumour, which had been discovered by her medical attendant eight years previously. An operation was proposed, but she refused, because the swelling gave her no pain. But after an attack of influenza the swelling became larger, painful and immovable. High fever and great weakness set in. There was no pus in the urine. Diagnosis : Infected closed hydronephrosis. At the operation, the perirenal tissue was found to be already infiltrated with pus, and pints of streptococcal pus issued forth from the renal sac. The subsequent course of the disease was marked by suppurative pleurisy of the same side.

(2) **Open Hydronephrosis.**—This is distinguished from the closed variety by the fact that, within certain limits, its volume is variable. It may be subject to an ascending infection along the urinary tract, which cannot occur to a closed hydronephrosis, and in this event the urine will contain pus, either temporarily or permanently.

(3) From the point of view of diagnosis, **intermittent hydro-**

nephrosis is the most interesting form. It depends upon congenital anomalies in connection with the renal pelvis or ureter, or upon the results of a movable kidney. This latter cause acts most frequently on the right side and in women.

The patient, who is either in perfect health or has been suffering from a dull ache in the loins, is seized with severe pain in one kidney—pain which radiates to the inguinal region, the genitals and the thigh. Sometimes the picture is completed by vomiting, great pallor, a collapsed pulse and cold sweats. If the hypochondrium is examined at this time it will be found to be occupied by a swelling varying in size from a fist to a man's head. This swelling is sometimes very difficult to feel, because of the reflex rigidity of the muscles. The symptoms persist for a few hours, rarely more than a day, and then subside after the profuse micturition of clear urine, which occasionally also contains blood. Sometimes the emptying of the kidney is delayed, especially if the sac is large; in other cases the kidneys do not return to their normal volume between the attacks—*remittent hydronephrosis.* This may merge into the chronic open variety, and, finally, into the acute variety. On examining a case of pure intermittent hydronephrosis during a free interval nothing abnormal is found, except perhaps a movable kidney. The diagnosis may be made in some cases from the fact that a swelling is to be felt in one side of the abdomen during the attacks and that they end with the abundant evacuation of clear urine, sometimes also containing blood. If, however, these indications are not present, one must wait until the next attack occurs. It is impossible to miss the diagnosis during the attack itself, at any rate, if the hydronephrosis has attained any definite size. But in the early stages before the tumour has reached the size of a fist the muscular rigidity may render its detection very difficult. The diagnosis will then lie between renal colic, biliary colic and even appendicitis, the last because the pain radiates downwards. But the localization of the pain on pressure and of the muscular rigidity in the lumbar region is decisive against biliary colic or appendicitis. Nevertheless there are cases wherein the question of an attack of gall-stones must be left in suspense.

If the hydronephrosis has become infected more or less pus will be found in the urine during the free intervals, and signs of infection will be present in addition to those due to the retention, viz., fever, rigors, dry tongue. The longer the disease lasts the more serious becomes the condition of the patient. Cystitis follows the hydronephrosis, and the other kidney is involved by an ascending infection. The disease finally terminates in uræmia, with or without the secondary development of stones.

It is important to recognize the cause of the hydronephrosis. If red blood cells can be demonstrated in the centrifugalized urine between the attacks, if these cells increase after physical exercise, and if the urine contains sand, or crystals which can be detected on microscopic examination, the cause is probably a *stone*. But a small stone in the ureter will fail to yield these signs. A decision can be arrived at by means of a good skiagram, wherein the shadow of the ureteral catheter should coincide with the shadow on the skiagram of a stone.

Tuberculosis is indicated by the discovery of tubercle bacilli, or by the successful inoculation of a guinea-pig. This form of tuberculosis occasionally runs its course under the guise of an ordinary intermittent hydronephrosis.

But if the investigations leading to the discovery of a stone or of tubercle be fruitless, we must assume that the hydronephrosis is due to kinking of the ureter. This is sometimes caused by a congenitally abnormal insertion of the latter.

Injury may lead to hydronephrosis of the three varieties described above—by scarring or kinking of the ureter, or by compression thereof. This possibility should be considered in taking the history of a case, even if the patient's account does not point to it.

CHAPTER LXIV.

IDIOPATHIC SUPPURATION IN THE RENAL PELVIS AND KIDNEY.

URINE which persistently contains pus, ascertained by the previously explained methods to be coming either entirely or partially from the kidneys, always raises the question whether the suppuration is an independent process, or whether it is a consequence of some antecedent condition, such as hydronephrosis, stone in the kidney, tumour or tuberculosis.

We purposely avoid the terms "primary" and "secondary," because the manner in which they are generally used is liable to cause misunderstanding.

An infection of the kidney is *primary* when the cause is introduced directly from without, and the kidney constitutes the first seat of attack. The same term is applicable if the causal organism has produced no pathological change at the point of entry, but has reached the blood, and thence has become deposited in the kidney.

The suppuration is *secondary* if the kidney is not the first organ to be attacked; for instance, if a cystitis has preceded the pyelitis (*urogenous infection*); or if the kidney infection which has taken place is *metastatic*, by way of the blood-stream, and has arisen from some anatomically demonstrable primary focus.

If, however, the renal suppuration arises and remains within *the kidney itself* without the aid of any other morbid condition, it should be termed *idiopathic*. If it has followed any serious pathological change in the organ, such as tuberculosis, stone or tumour, we apply to it the term *complication* or *sequela*.

After all, it is only a matter of words, and we might, with equal justice, insist on some other nomenclature. The main point is, however, always to use the same expression for the same pathological process. No scheme has the advantage of being strictly maintained throughout, and there are always some processes which might properly be classified in various positions. For instance, pyelitis, which is caused by urinary obstruction in enlarged prostate, may be looked upon either as a sequela or as an independent suppurative process. The point turns upon the amount of predisposing influence we will allow before we abandon the conception of "independent suppuration." There is really no definite border line, and if we have, for example, fixed it on the other side of prostatic hypertrophy it is solely for the purpose of classification.

Tuberculosis is certainly a form of "idiopathic" suppuration, but as we are discussing the causes of *acute* suppuration, and as urogenital tuberculosis is clinically an independent disease, we will not include it here, but will devote a special chapter to it.

When the purulent infection is merely a *sequela* or a *complication* of an existing renal disease, it will usually have been preceded by a stage wherein the symptoms of the primary disease have been clearly manifested, especially in the case of hydronephrosis, often also in the case of stone, and occasionally, though less definitely, in tumours. If not, we must rely upon the physical examination for the differential diagnosis. An abscess sac as large as an infant's head, or larger, does not indicate idiopathic suppuration, but has probably originated in an old hydronephrosis. A large irregular tumour indicates a new growth. A constant, although slight, admixture of blood with the urine, in a case of renal suppuration suggests stone or tuberculosis; greater hæmorrhage might also be due to stone. But if nothing of this kind can be discovered, we may regard the suppuration as "idiopathic" in the above limited sense, and we must search for the conditions which might account for it. Boils, erysipelas, sore throat and gonorrhœa—in an ascending scale—are the main distributors of infection. Pregnancy and the puerperium with its deficient micturition are causes of slight urinary stagnation, and in these the midwife often provides the infective material.

It is only rarely that *bacteriological examination* will elucidate the matter—for instance, if it demonstrates the presence of *Staphylococcus aureus*, the pneumococcus or the typhoid bacillus. Streptococci and colon bacilli are, however, such frequent denizens of diseased urinary passages that we cannot draw any definite conclusion from their presence.

The gonococcus is hardly ever found. This is, as a rule, only the first link in the chain of infection, and is followed by cystitis and pyelitis due to mixed infection—these are the second and third links. I have seen vesical stone and renal stone after gonorrhœa—constituting fourth and fifth links, and in a similar case also cancer—a sixth link. Of course, a "harmless" gonorrhœa does not always proceed as far as this. More frequently a stricture is interposed in the chain of sequelæ, which may lead, in after years, to an ascending urinary infection.

It is important to ascertain whether such an infection is *unilateral* or *bilateral*. The etiology may help to decide this point, for the pyelitis of pregnancy and post-gonorrhœal pyelitis are usually unilateral —at any rate at first—whereas in prostatic patients it is generally bilateral. If the disease is metastatic in origin, it may be on one side or on both. If the patient states that his pain is sometimes in one loin and sometimes in the other, the pyelitis is very probably bilateral. Valuable diagnostic points are afforded by the demonstration, by palpation, of tenderness on pressure, by reflex muscular rigidity and sometimes by enlargement of the kidney. Frequently, however, the kidney is neither tender nor enlarged when in a condition of pyelitis. It is then advisable to attempt to palpate the ureters, either *per vaginam* or *per rectum*. If they can both be felt as corps, then both kidneys are diseased (Garré). A final decision may be arrived at by means of the cystoscope and the separation of the urine.

The *anatomical diagnosis* presents the most difficult problem, and it involves the differentiation of the following conditions—simple catarrh of the renal pelvis, i.e., pyelitis; simultaneous disease of the renal parenchyma, i.e., pyelonephritis; and, finally, disease exclusively confined to the parenchyma, i.e., simple or multiple abscess of the kidney.

Both pyelitis and pyelonephritis are very frequently due to ascending infection, but they are of a hæmatogenous nature, more often than was previously supposed. Pain on pressure and enlargement of the kidney, as well as acute symptoms, only occur in either condition during the stage of retention (pyonephrosis). The involvement of the renal tissue is recognized by the fact that the albumin is proportionately too high in comparison with the amount of pus, and also occasionally by the presence of cylindrical casts. But even in the absence of these signs, experience tells us that the renal tissue does not remain healthy after a long-continued pyelitis.

Renal abscess, whether single or multiple, is the result of metastasis, and is accordingly a pure infection, in contrast to an ascending infection, which is usually of a mixed character. It is easily overlooked, because there is no pus in the urine at first, and nothing but the fever and lumbar pain indicate its presence. There is no difficulty in distinguishing it from pyelitis. Bilateral renal abscess is very rare, but this condition is indicated by tenderness on pressure in both lumbar regions. It is, however, quite impossible to decide whether the abscess is a large single one, or a collection of numerous miliary abscesses. If the illness is severe and quite recent, the former condition is suggested ; if the illness is less severe and more prolonged in its course, probably the latter condition is present.

Renal abscess is distinguished from perinephritis by the slighter tension of the lumbar muscles. The differentiation is important, because small renal abscesses often heal spontaneously, whereas perinephritis always demands surgical interference. But in some cases the differential diagnosis is impossible, and surgical intervention must depend upon the spontaneous disappearance of symptoms after a few days or their persistence.

It should be repeated, that the most usual, if not invariable, origin of perinephritis is in an abscess of the renal cortex, which has not been recognized clinically.

In rare cases a renal abscess sets up so little local reaction that one is tempted at first to think of a new growth.

A man, aged 60, came for advice regarding an indefinite pain in the left loin. Examination showed that there was in this region a moderately tender, somewhat nodular and rather movable swelling. There was no pus in the urine, but a quantity of sugar. There was moderate pyrexia, and the patient died in a few days from pyæmia. At the autopsy it was seen that what appeared to be a renal tumour was really the kidney, infiltrated by a well-encapsuled abscess.

We should always think of a *renal infarct*, in every case of acute renal disease of an obscure nature.

The following case will show how one may occasionally be misled:—

A middle-aged man was suffering from an old neglected gonorrhœal stricture, cystitis and intermittent attacks of "urinary fever." He received some casual treatment from a chemist, but suddenly became ill with severe septic symptoms, spontaneous pain and tenderness on pressure over the right kidney, which, however, was not enlarged. The left kidney was slightly tender, but also of normal size. The urine was almost free from pus, which was attributed to retention within the pelvis of the right kidney. But nephrotomy on the right side showed that there was neither retention nor pus. The septic symptoms increased and the patient soon died. The autopsy revealed recent multiple non-infective *infarcts* in both kidneys, due to vegetative endocarditis.

CHAPTER LXV.

STONE IN THE KIDNEY AND URETER.

PRIMARY stone in the kidney plays a very small part in the renal pathology of some countries, while in others it is one of the most frequent of maladies. In some countries stone has even become quite a disease of children.

We distinguish primary from secondary stones, and we separate the former into a non-infected and a secondarily infected variety.

A.—PRIMARY STONE IN THE KIDNEY.

(1) NON-INFECTED STONE.

There are four important symptoms which establish the diagnosis of nephrolithiasis, *viz.:* (1) attacks of renal colic; (2) dull aching pain in one loin during the intervals; (3) hæmaturia, which may be very slight, but increases with movement; (4) presence of gravel or larger concretions in the urine.

Renal colic runs a similar course in stone, as in intermittent hydronephrosis, with the one difference, that the swelling due to the retention is not so large, and that, in consequence, the urine which is passed after the attack is over is smaller in quantity and contains less blood. But, nevertheless, a temporary reflex polyuria may occur. The radiation of the pain into the inguinal region may resemble an appendicitis, and the pain on pressure under the liver may lead to confusion with gall-stones. Spontaneous pain at the testicle is very suggestive of renal stone. The testicle is very often tender on pressure.

Dull aching in the loin is a symptom of great significance, but must be employed with discretion, especially if there is any doubt about the diagnosis of renal disease, as against biliary colic, for example.

We have already dwelt in detail on *hæmaturia* and *gravel* in the general section, and we have seen that the former symptom is more constant, but less in amount, than in cases of tumour. Recent observers have laid great stress on the fact that traces of blood in the sediment of the urine, increasing in amount after movement, may, for a long time be the only sign of stone in the kidney. There may be no renal colic, and the dull pain in the loin may be entirely absent, as occurs with stones which remain latent for years. These cases do not, as a rule, seek any advice. The traces of blood in the urine are discovered quite accidentally; for instance, if the patient consults his doctor for indigestion or some other condition which apparently has no connection with the kidney. The dull pain in the loin is often put down to lumbago or rheumatism. If there be no pain at all in the case of a small stone situated in the ureter and causing no hæmaturia, a diagnosis is absolutely impossible during the quiet interval.

A *skiagram*, taken by an expert and interpreted by an experienced observer, should have the last word in all diagnostic difficulties connected with stone in the kidney.

The diagnosis of stone in the kidney by X-rays is not easy, because stones composed of uric acid and of urates only cast a faint shadow, which in fat patients is hardly perceptible. But the experience in districts wherein this disease is common has been that even these stones often contain so much lime that they may be rendered visible on the plate, under proper conditions. No decisive conclusion should, however, be made until every doubtful appearance has been examined on several impressions.

The *composition* of the stone is important from a therapeutic aspect.

If the patient is a gouty subject, or if he comes from a gouty stock the stone probably consists of uric acid or of urates, even in children. If crystals or gravel be found in the urine, we shall obtain some guidance from their microscopic and chemical examination, the details of which have already been referred to.

(2) INFECTED STONE.

In addition to the symptoms of aseptic nephrolithiasis, those of infection are present: pus in the urine; fever and rigors where there is retention. The correct diagnosis is very often missed, and the condition appears to be either an independent pyelitis or tubercle. It must be remembered that when the ureter is temporarily obstructed the urine may be quite normal, because it all comes from the healthy kidney, and it is, therefore, always necessary to examine the urine during an interval when the ureter is not blocked.

B.—SECONDARY STONE IN THE KIDNEY.

Most renal concretions which occur in non-calculous districts are secondary in character. Their symptoms consist of those of the underlying suppurative disease, to which are superadded the symptoms of stone. The clinical picture is practically the same as that of an infected primary stone, but the history is different. In the present instance, suppuration precedes the stone; in the other instance the stone precedes the suppuration. This consideration will indicate the difficulties which may arise in differential diagnosis, as well as their solution, and we will therefore not repeat what has already been said.

Chemically, all secondary stones consist of earthy phosphates and carbonates. Their formation is recognised in the urine by its ammoniacal smell and the abundance of triple phosphates. Röntgen-rays reveal these stones very clearly, because of their rich calcium content.

The difficulty which may exist in differentiating between acute cholecystitis, appendicitis, and an infected renal calculus is well illustrated by the case quoted in the section on acute cholecystitis (page 337).

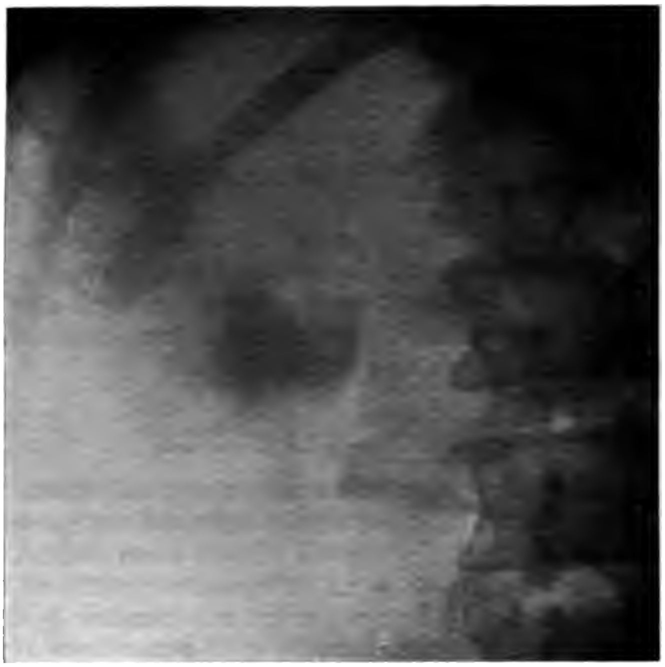


FIG. 256.—Skiagram of stone in kidney.

The following observation is also noteworthy. A man aged 55, had been suffering for some years from attacks of pain, which some physicians attributed to gastric ulcer and others to stone in the kidney. A careful analysis of the individual attacks showed that they were divisible into two separate types. The one type was chiefly characterized by anæmia and collapse, associated with intestinal hæmorrhage; the other type consisted of pain in the left lumbar region and pyrexia. Chronic cystitis was also present. The inference was that both ulcer and stone were present. A skiagram showed that there was a deep notch in the stomach, indicating an ulcer, and also that there was a stone in the kidney, illustrated in fig. 256.

CHAPTER LXVI.

TUMOURS OF THE KIDNEY.

TUMOURS of the kidney, as long as they are not infected, are recognized by three symptoms: (1) *hæmorrhage;* (2) local, and especially radiating *pain;* and (3) *swelling.* The predominance of the one symptom or of the other depends upon the position or manner of growth of the tumour.

Hæmorrhage is absent in very few cases only. It is much more profuse, but also much more irregular, than in the case of stone. If the hæmorrhage is very pronounced, the clots may obstruct the ureter temporarily, causing genuine renal colic, which must, however, be distinguished from the persistent radiating neuralgic pain. It is noteworthy that such hæmorrhages may continue for years. The radiating pain does not occur until a late stage, so that the hæmorrhage, with or without renal colic, may be for years the only symptom of a small stationary tumour of the kidney, which cannot be felt. It is only by the cystoscope, showing that the blood comes from one side, that such a case can be distinguished from chronic hæmorrhagic nephritis, which may exist for a long time without albumin or cylindrical casts. If the cystoscope does not yield a decisive result, an exploratory incision must be resorted to, and even then it is not always easy to discover the source of the bleeding.

If the hæmorrhage is very profuse and persistent, one should think of the possibility of a new growth from the *pelvis of the kidney,* especially if no appreciable swelling can be demonstrated, or if the examiner can feel a hæmatoma which is causing extreme distension of the renal pelvis (Israel).

Persistent, local, and radiating neuralgic **pain** merely tells us that the tumour is malignant, and that operation will probably be fruitless. If such pain is unaccompanied by any other symptom, the unfortunate diagnosis of lumbago is often ascribed to it, but this is an error which tumours of the kidney share with all painful diseases of this region.

If the **tumour** is the most striking symptom we must first decide whether it is really connected with the kidney. If the hæmaturia is present at the same time the matter is clear. I have, however, several times seen a hypernephroma diagnosed as a tumour of the spleen, because the patient had lived in a malarial region or had actually suffered from malaria. But if the urine is normal, and the cystoscope shows that it comes from both kidneys in equal amounts, then we should think of a liver or gall-bladder tumour on the right side,

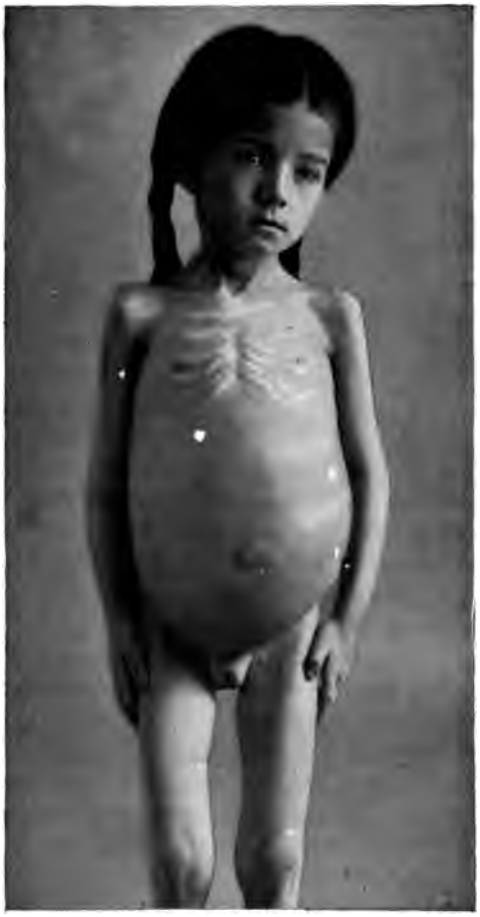


FIG. 257.—Sarcoma of the left kidney.

a splenic tumour on the left side, and a tumour of the large intestine on either side. If the tumour is of unusual size, an ovarian cyst should be thought of. There is, however, one sign which distinguishes a renal tumour from all of these, the fact that, on bi-manual palpation, it can be felt most distinctly from behind, in the angle between the spine and the twelfth rib. A tumour of the intestine would generally cause some intestinal disturbance. A tumour of the gall-bladder has its own special previous history, and a swelling of the spleen betrays itself by the sharp anterior border, which can usually be felt quite easily. It is only in the case of an irregular round tumour of the spleen that serious difficulty can arise. An ovarian tumour is recognized by the circumstance that the large bowel runs over the new growth, as can be shown by a skiagram. It takes the same course in the case of new growth or hydronephrosis of an ectopic kidney, but on the left side, the large bowel has the same relation to an enlarged kidney which is normally situated. Soft tumours of the fatty capsule of the kidney (lipomata, fibromata, myxo-sarcomata) which may attain a large size, have hitherto only been diagnosed at the operation. They may cause the most extraordinary displacements of the viscera.

A cystoscopic examination may enable a decisive opinion to be given either for or against a renal tumour (persistent absence of urinary flow from one side). The collargol method is also of value if this procedure may be recommended unreservedly.

Having decided that a tumour of the kidney is present, we must determine whether it is a *retention tumour*—hydronephrosis or pyonephrosis—or whether it is a genuine new growth. This point is usually elucidated by the history and the condition of the urine. The consistence of the tumour may be very misleading, because a sarcoma may feel just as elastic as a hydronephrosis. One would only think of a *hydatid* in districts where this disease is rife, but localization in the kidney is always very rare. Exploratory puncture, which is occasionally recommended, is just as inadvisable here as it is in the case of the liver, and the serum test is still unreliable.

It is difficult, or quite impossible, to infer anything about the histological characters of a renal tumour diagnosed by its clinical signs. There are of course cases wherein the uneven surface and the slight mobility of the tumour stamps it conclusively as malignant. But we often remain in doubt because, as we have seen, the hæmorrhage from malignant tumours even may persist for years. It is only rarely that external circumstances permit us to make a definite diagnosis on clinical grounds. This, however, applies to the tumours of childhood, which experience shows are either pure sarcomata or mixed sarcomatous tumours. If there is no hæmorrhage one would be obliged to think of hypernephroma, which so often arises in the cortex. If the tumour is bilateral, round and uneven, without hæmorrhage or suppuration, and if the discomfort is limited to a dull pain with occasional renal colic, the only possible diagnosis is congenital cystic kidney, especially if there is also enlargement of the liver (cystic liver).

From the point of view of treatment the precise nature of a new growth of the kidney is not of much importance, because *as soon as a renal tumour has been demonstrated it must be removed, unless it is too late.*

Cystic kidneys are an exception to this rule; these are only to be removed in the rare instances wherein it is certain that they are unilateral, and then only if they cause sufficient trouble to justify the operation.

Finally, it must be remembered that renal tumours do not always occupy their normal position, resembling in this respect the kidneys themselves. Tumours in floating kidneys are not uncommon, and new growths may develop in congenitally displaced kidneys, which usually lie at the level of the pelvic inlet.

I once removed a hypernephroma of such a pelvic kidney. Its true nature was only recognized at the operation by the fact that the kidney was absent from its normal position. The clinical diagnosis fluctuated between a solid ovarian and a renal tumour.

CHAPTER LXVII.

TUBERCULOSIS OF THE URINARY PASSAGES.

ALTHOUGH the prognosis of tubercle of the urinary passages is favourable in its early stage, the prospect is one of the gloomiest when the disease is advanced. Unfortunately the early stage is often overlooked, because it does not declare itself very definitely. Every disturbance of the urinary organs of gradual onset should therefore make one think of tubercle, and decide for or against it, instead of allowing the patient to go about for months, with the vague diagnosis of vesical catarrh, vesical irritation, or simply "neurasthenia," and with unsystematic or so-called "symptomatic" treatment. The first symptom is usually a certain amount of tenesmus. The patient notices, as the most striking objective sign of this change, that he has even to get out of bed once or twice during the night. This differentiates him from a neurasthenic, who may micturate very frequently during the day, but is not disturbed at night. At this stage, the naked eye can detect nothing wrong with the urine. A careful examination may, however, reveal traces of albumen and a slight deposit of pus cells, epithelial cells and isolated blood cells, in the centrifugalized sediment. There are usually no bacteria present, nor even tubercle bacilli. This condition of the urine absolutely excludes a simple neurasthenia, in which one finds phosphates, carbonates, calcium oxalate and occasionally one or two seminal threads. Such a urine supports the view of some organic disease, and the patient should be thoroughly examined, when it is most likely that some old scars of glands or an apical catarrh will be met with. At this stage, palpation of the kidneys will not usually yield any result, but a tender spot will often be found in the prostate, especially on its superior surface, and occasionally also a nodule in the epididymis. But if none of these points affords a positive indication of tubercle, it will be necessary to inoculate a guinea-pig with an adequate amount of the scanty sediment. It may then be found that, although the recent examination of the urine revealed no tubercle bacilli, the animal becomes tubercular within four to eight weeks. In this way, an *early diagnosis* of tubercle may be made, and appropriate treatment started.

Recent researches have shown that tubercular persons may excrete isolated tubercle bacilli, even though their kidneys are unaffected. We must therefore not assume the presence of local disease of the urinary organs unless the bacilli are found frequently in large numbers, and the urine contains traces of pus—and if the kidneys are in question, it must be shown that their function is definitely disturbed. The case is *suspicious*, if there is a striking unilateral polyuria; but it must not be forgotten that the very passage of a ureteral catheter sometimes stimulates a healthy kidney to a temporary increase of excretion.

Having thus detected that the urinary system as a whole is affected with tubercular disease, we must now search for its *point of origin*. Clinical experience is accumulating proof that the disease starts in the *kidneys*, or in one of them. Spontaneous pain, local tenderness on pressure, slight rigidity of the lumbar muscles, perhaps also some demonstrable enlargement of the organ, and, occasionally, thickening of the ureter, felt through the rectum or vagina, will show which kidney is affected. If none of these indications is present, the practitioner will have done his duty by referring the patient to the surgeon as a case of "urinary tuberculosis." If however the practitioner is able to avail himself of a cystoscope, the inspection of the two ureteral openings will show which is the diseased side.

On the affected side the margins of the ureteral openings are reddened and swollen, whilst the orifice itself is often strikingly gaping. Around it, there may be a few tubercles or small ulcers. In somewhat more advanced cases the urine which escapes is distinctly turbid.

This examination is completed by separating the urine within the bladder. If this procedure is repeated several times it yields most useful results. Catheterization of the ureters is even more reliable, but demands more skill. The question of operation and its method must then be left to the surgeon. The decision depends upon the presence of a healthy, or at least of an adequately functional and non-tubercular kidney on the other side.

In this connection, it must be emphasized that all these manipulations must be carried out with special care and with asepsis. An instrument should not be introduced into a tubercular bladder, which is not affected by a mixed infection, or only slightly so, unless some definite diagnostic information is anticipated from the procedure, or unless it is done for a definite therapeutic purpose. Whenever an examination is made with a sound, cystoscope, separator or ureteral catheter, it should have been preceded by the administration of a urinary antiseptic such as urotropine, or at any rate be followed by it.

The differential diagnosis of the early stage of urinary tuberculosis varies with the initial symptoms. If *hæmorrhage* is the predominant sign, as occurs in exceptional cases, one thinks of *new growth*. If the early stage is characterized by *renal colic*, there is a possibility of confusion with *stone in the kidney*, *intermittent hydronephrosis*, and even with *appendicitis*.

I once saw a patient whose first symptom of renal tuberculosis was an attack which was regarded by most experienced surgeons and physcians as one of appendicitis. The correct interpretation of the condition was not forthcoming until the urine was examined.

If there are no striking bladder symptoms, but *lumbar pain* is present, the diagnosis of rheumatism or lumbago usually suffices, if the patient thinks it at all necessary to consult a doctor.

The practical question which most concerns this matter, is the advisability of nephrectomy in the early stage of renal tuberculosis. It is necessary to have a very clear conception of what is meant by an early stage. A patient is not necessarily in an early stage of renal tuberculosis because the condition has just been diagnosed. Indeed it may then be in a very advanced stage. We can only be confident that the stage is early, if a comparative investigation shows that the parenchyma of the kidney has not suffered much disturbance in function.

The later stages of the disease also present their problems.

There are some cases in which we must assume an original focus of tubercle in the kidney, but wherein the bladder symptoms are so predominant that they attract all the attention. Generally, it is the *vesical tenesmus* which is so conspicuous and causes the patient most torture.

This symptom usually indicates an accompanying tuberculosis of the bladder, but it may be due to reflex causes from the kidney. If the pain is intense, it suggests the secondary formation of stone.

The later stages of urogenital tuberculosis are often mainly characterized by this *formation of stone*, with all the symptoms of secondary infected renal and vesical stones, with renal colic, fever and rigors.

The liability to secondary stone formation starts, as we have seen, at the time when the urine, which was originally acid, becomes alkaline owing to mixed infection. This affords us a reliable means of recognizing whether the renal colic which is present is due to stone or not. Sometimes, however, this diagnosis is facilitated by the passage of small concretions.

It is important to recognize this secondary stone formation early, because the removal of these stones will give great relief even in cases where, owing to the tubercle affecting both sides, there is no prospect of complete cure.

In a case of bilateral renal tuberculosis, where radical operation was impossible, I removed a large number of stones in two sittings from the right pelvis, one stone from the right ureter, and a large stone from the bladder. In this way great relief was afforded to the patient for about a year, although, of course, it did not prevent the eventual onset of uræmia.

On the other hand, we must not attribute cases of severe chronic tenesmus to tubercle, without careful examination. Vesical stones may cause no other symptom but tenesmus, even without any tubercle.

We must also refer here to **perinephritis**—a not infrequent complication of renal tuberculosis. It occurs in two forms, which are easily distinguishable clinically. One form consists of a sharply-defined abscess which, without any marked symptoms, tracks downwards or bursts through, in the lumbar region. Cultures made from the pus are sterile, but an inoculated guinea-pig becomes tubercular. This form constitutes the purely tubercular stage of the disease, wherein the focus in the kidney bursts externally just as a focus in bone leads to the development of a cold abscess. In the other form the perinephritis manifests itself by acute symptoms, fever, rigors, and severe pain, and instead of a circumscribed abscess we have a phlegmon. This process is really a mixed infection, and its intensity depends upon the virulence of the streptococci or colon bacilli, &c., which take part in it. We must, therefore, not discard the possibility of a tubercular origin for the renal malady because of the acute character of the perinephritis. Tubercle does not usually penetrate lower than the sphincter vesicæ, but may cause changes at that site which may be mistaken for late sequelæ of gonorrhœa, if there be a history of that disorder and a bacteriological examination of the urine is neglected. Tuberculosis of the genital organs is not dealt with here, having already been discussed in a previous chapter.

CHAPTER LXVIII.

STONE IN THE BLADDER.

We differentiate between aseptic and infected stones, just as in the case of renal stones.

(1) Three symptoms point, as already seen in Chapter LX, to **non-infected stones in the bladder**: irregular and varying disturbances of micturition, not affected by changes in posture, vesical tenesmus and hæmorrhages.

Obstruction at the neck of the bladder by a valve action is very significant of stone, but this is frequently absent, especially when the stone is large or within a diverticulum—in the latter circumstance the stone is no longer aseptic.

Tenesmus is the result of direct mechanical irritation, and is very marked in the case of rough oxalate concretions. The tenesmus is aggravated by any vibration of the body, especially by riding.

A patient of mine with an oxalate calculus always selected the back platform of a last carriage when on a railway journey, so that he could empty his bladder from there, as the need became urgent.

The *hæmorrhage* is usually very moderate, just as it is in renal stone, and in contrast to the hæmorrhage of new growths.

Whenever a vesical stone is suspected we should investigate the history in regard to gout, and also search for any indications of renal stone. Most vesical stones originate in the kidney, but become large in the bladder. We then examine the urine, or the sediment obtained from a large quantity thereof, for crystals or small concretions. Then, after the bowel is emptied, a bi-manual examination of the bladder is made with one finger either in the rectum or vagina and the other hand on the abdomen. Large stones may then be felt quite easily. We may next proceed to use the sound. This examination must be conducted with great patience and with the bladder in varying degrees of fullness, if the stone cannot be felt on the first attempt. The sound also gives information as to the smoothness or roughness of the surface of the stone; in some cases we may be able to tell its size. The most reliable results in regard to the number and size of stones are afforded by the cystoscope and an X-ray examination (fig. 258); for the latter it is necessary that the bowel should have been previously emptied by an enema.

The patient must be undressed for this examination, otherwise one runs the risk of opening the bladder for stone when in reality the shadow is due to a trousers button—an incident which has actually happened.

An aseptic stone in the bladder may be mistaken for—

(*a*) *Tumour of the bladder*, especially for a *polypus* at the neck of the bladder, causing obstruction by valve action, and tenesmus—a very rare occurrence. Such a condition should be thought of, if the sound and the skiagram yield negative results. Under these circumstances a cystoscopy is decisive.

(*b*) *Tubercle* which still remains aseptic, in cases where there is not much admixture of pus in the urine.

(2) **Infected Stone in Bladder.**—If a bladder containing a stone become infected spontaneously or through catheterization, the previous symptoms are supplemented by *suppuration* and by an increase in the tenesmus. The other symptoms remain *in statu quo*. The case is then very liable to be mistaken for some form of cystitis—especially of a tubercular nature.

Secondary stones in the bladder resemble, in their behaviour, infected primary stones. They are found as a result of suppurative infection of the urinary passages and of alkaline, generally ammoniacal, decomposition of the urine. Their nucleus is often some foreign body, such as a piece of catheter, a hairpin, a nail, &c. The history of these secondary stones differs from that of the infected primary stones, because in the former the infection either with or without a foreign body precedes the stone, whereas in the latter the stone precedes the infection—just as in the case of renal stones.

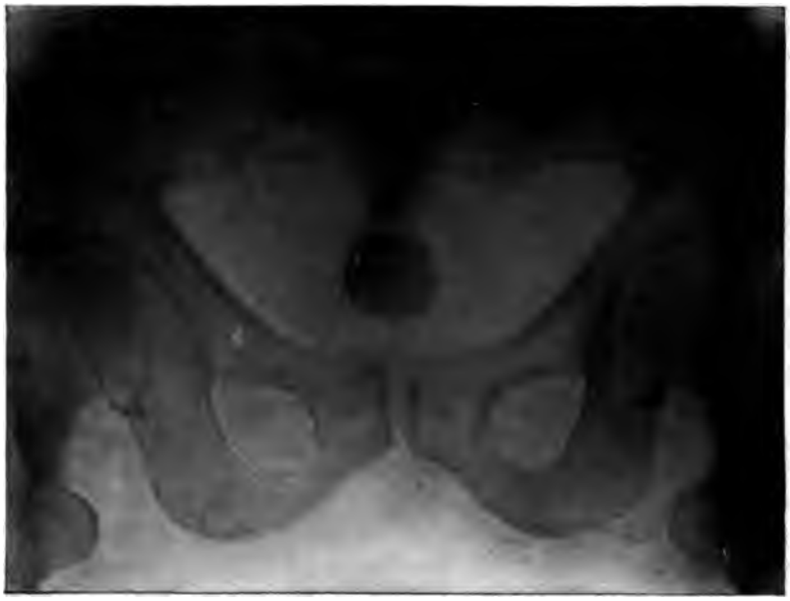


FIG. 258.—Skiagram of stone in bladder.

The *original malady* is often an old gonorrhœal or puerperal cystitis or one due to spinal paralysis. In other cases it may arise from urinary infection after an enlarged prostate ; sometimes tubercle is the underlying cause. In rare cases it is a congenital diverticulum which has led to local congestion of urine, to the occurrence of a spontaneous infection, and, eventually, to the formation of stone.

It happens sometimes that there is no interference with micturition, which is due to the circumstance that the stones may be firmly fixed in *diverticula*, or that they may be too large to act as ball valves. These cases manifest themselves by an extremely agonizing vesical tenesmus, which cannot possibly be relieved, and which eventually resembles incontinence. Stones within diverticula are easily missed by the sound, and this makes their diagnosis all the more difficult. Cystoscopy sometimes fails to yield the desired result in these cases, because of the diminution in the capacity of the bladder, consequent upon the constant strangury ; but these stones can always be demonstrated by a skiagram.

CHAPTER LXIX.

CYSTITIS.

In devoting a few lines, in addition to what has already been said, to the diagnosis of cystitis, the most important consideration to emphasize is, that this diagnosis is made too often. There is a tendency to be content with the assumption that there is a catarrh of the bladder, instead of ascertaining the origin of the trouble. One who always diagnoses cystitis when there is pus in the urine and strangury is present, will miss most cases of prostatic abscess, uro-genital tuberculosis, pyelitis and infected stones. It matters not that there is some catarrh of the bladder present in most of these cases, because it is not the incidental malady, but the fundamental disease which has to be recognized and treated. Even if the cystitis should be the primary and original disease, we must not be content with this diagnosis, but must, if the disease does not rapidly recover, follow up the secondary changes—pyelitis and stone formation—which cause the trouble to become chronic.

The etiology renders the principal assistance in the diagnosis of a primary cystitis. Catarrh of the bladder never originates "of itself," through some constantly prevalent infection, like a cold. It is always due to some definitely demonstrable cause—introduction of some infective organisms from the kidney or from without, on the one hand, and such predisposing conditions as *urinary congestion*, *injuries* and the presence of *foreign bodies* on the other hand. The more virulent the organisms, the less individual predisposition is required to evoke an attack, and vice versa.

The puerperal bladder, with its dilatory powers of micturition affords a well-known example of the influence of even slight congestion.

The following cases illustrate the significance of injuries to the mucous membrane:—

A healthy young woman, in whom gonorrhœa could be excluded, was suddenly seized with severe cystitis, and the passage of offensive urine. The history, which was elicited with difficulty, showed that in using a vaginal injection prescribed by her physician, she had by mistake introduced the tube into the urethra, and this had severely injured the neck of the bladder. The bladder would have soon got rid of the infection if it had not been for the injury.

The same applies to foreign bodies. A practitioner had the misfortune to leave a piece of a Nélaton's catheter in the bladder of an elderly female. Severe cystitis with ammoniacal decomposition rapidly supervened, and I found the foreign body on examination, fourteen days subsequently, completely encrusted with triple phosphates. This cystitis did not signify that the practitioner was not cleanly in his procedure—he was well acquainted with the theory and practice of asepsis—but simply that a slight, and perhaps unavoidable, infection sufficed to provoke a severe catarrh of the bladder in the presence of a foreign body.

I say "perhaps unavoidable," because we know that even a healthy urethra harbours micro-organisms which we may introduce into the bladder, even with the most careful asepsis. The reason that the passage of a catheter does not more frequently lead to an infection than it does, is because the normal bladder is able to dispose of most micro-organisms quite easily. For the same reason, cystitis occurs so rarely when micro-organisms are excreted from the kidneys into the bladder. Indeed, the very existence of bacilluria shows that micro-organisms may continue to develop in the urine without injuring the healthy bladder.

If a large amount of pus suddenly appears from the bladder without any severe signs of irritation of this organ, the most probable cause is rupture of a perivesical abscess into it; the symptoms of the original disease—generally appendicitis—easily allow the diagnosis to be made.

Our modern period of operations for hernia has often witnessed infected sunken sutures and ligatures make their way into the bladder instead of externally, and thus cause cystitis, or the secondary formation of stones.

If nothing points to the cause of the infection of the bladder, and if its progress from the start has been very gradual, we shall rarely err in ascribing it to tubercle. Nevertheless we often find, even in children and young people, cases of obstinate cystitis, which soon lead to deposits of lime, whose chronic character can only be explained by some general decrease in resistance, in the sense in which we have used the term scrofula, but which are not definitely tubercular. (Chapter XXIV.)

CHAPTER LXX.

TUMOURS OF THE BLADDER.

THE clinical picture presented by a tumour of the mucous membrane is quite different from that of tumour of the muscular coat.

(1) TUMOURS OF THE MUCOUS MEMBRANE OF THE BLADDER.

The chief symptom of these tumours, like those of the kidney, is irregular hæmorrhage, which, having once started, becomes very severe and may cause profound anæmia, before any other signs appear.

All the other symptoms depend upon the position and form of the tumour, and upon complications. Thus, if the growth is near the neck of the bladder *strangury and retention* may occur; if it is of polypoid shape the symptoms are very *variable*; if it soon begins to invade the surrounding parts, *radiating pains* are felt in the region of the pelvic nerves and the great sciatic; if it compresses a ureteral orifice *renal colic* is experienced, and *difficulty in defæcation* if it grows into the rectum. A growth situated at the vertex of the bladder will betray itself chiefly by increased strangury, in addition to hæmorrhage, but, nevertheless, this form is one which goes longest unrecognized. As soon as cystitis supervenes, and this rarely fails in growths from the mucous membrane, vesical tenesmus occurs in addition to hæmorrhage, and becomes predominant, whatever be the situation of the growth. This tenesmus increases if deposits or concretions of triple phosphates form.

Symptoms of bladder irritation occur very early in cases of so-called *anilin cancer* of the bladder.

The fact that long continued occupation with certain chemicals, (especially para-amidophenol) leads to the development of papilloma or of cancer in the urinary tract is of diagnostic importance. Theoretically, this is one of the most significant facts in the whole study of tumour formation, because it absolutely disposes of the one-sided view of the parasitic origin of tumours. It is also noteworthy that we are able to follow histologically the entire process, beginning with simple irritative proliferation of the epithelium, going on to papilloma and culminating in cancer or sarcoma.

These symptoms having suggested a tumour of the bladder, we must examine the urine for the narrow villous-like shreds, which may at once furnish a diagnosis, or for the greyish-red pieces of tissue which require microscopic examination to determine their nature.

We then palpate the bladder in the full and empty state, after the bowels have been emptied. Growths of the base of the bladder can be felt distinctly, either from the rectum or vagina—frequently, however, as a diffuse resistance rather than as a defined growth. New growths of the vertex of the bladder can be more easily reached from the abdomen, but always by bi-manual examination, even in the male sex. A fat patient, or one whose abdominal wall is unyielding, will require an anæsthetic.

If we feel a resistant circumscribed structure, it may even be a stone, which, if enclosed in a diverticulum, will be immovable. Examination with the sound and cystoscope will at once show whether a stone is or is not present. Care must be taken not to mistake the not infrequent incrustation upon a growth for a stone, and in using the cystoscope in a female the uterus projecting into the bladder must not be regarded as a tumour. If sufficient experience is brought to bear upon the interpretation of the cytoscopic appearance, it should be quite decisive in regard to the diagnosis of tumour. But sometimes the size of the growth and the smallness of the interior of the bladder prevent such an examination. In such circumstances, however, palpation can elucidate the condition, except in the case of a very soft papilloma. If the cystoscope does not exclude the diagnosis of stone, an X-ray examination should be made.

The question of the innocence or malignancy of the growth is less defined in this region than in any other, because the most frequent tumour of the mucous membrane—**papilloma**—is often clinically very much on the border line. A small villous tumour which has been removed quite early, may be innocent, but an extensive papilloma approximates very much to a malignant growth, owing to its tendency to spread at its edges and to recur. Papillomata which are apparently innocent at first, may eventually become **cancerous**, and definite cancers may originally have possessed all the external characters of papillomata. A growth which bleeds and feels hard must be regarded as cancer, without hesitation.

If nothing, or at most some indefinite resistance in the bladder region, is felt on palpation, we should think of a papilloma as most probably present. This may invest the whole bladder, without forming a large tumour. A cystoscopic examination is indispensable in such a case.

(2) TUMOURS IN THE MUSCULAR COAT OF THE BLADDER.

The conditions are quite different when a tumour arises in the muscular layer—**fibroma, myoma, sarcoma.** The tumour breaks down and the hæmorrhage starts, if at all, in a late stage, so that the disease is only recognized when its extension compromises the functions of the bladder by mechanical interference. If the new growth is on the posterior wall of the bladder, it may resemble a myoma of the uterus growing forward ; an operation alone can reveal the correct relations of the tumour.

A myoma of the uterus sometimes grows between this organ and the bladder, connected to the uterus by a narrow stalk only, displacing the muscular layer of the bladder to a very large extent, and may in fact cause its total disappearance. On the other hand, a fibroma or myoma arising in the wall of the bladder may invade the uterus to such an extent that it is only the absence of a pedicle connecting it to that organ which shows that it is independent thereof.

CHAPTER LXXI.

HYPERTROPHY, TUMOURS AND ABSCESS OF THE PROSTATE.

ALTHOUGH we have already touched upon diseases of the prostate, we will summarize once more the most important of them and amplify our previous remarks by a few points.

(1) HYPERTROPHY AND TUMOURS.

If an elderly man has constant trouble in emptying his bladder, although a large catheter can be introduced, we should at once think of enlargement of the prostate. Rectal examination will in most cases show that the organ is enlarged. If we can inspect the interior of the bladder, we will usually see two lateral swellings of somewhat unequal size at its entrance; sometimes only one eminence in the centre (clinically known as the middle lobe); occasionally a ringed-shaped pad-like projection is seen at the neck of the bladder.

Enlargement of the prostate does not usually affect the whole organ, nor even any special part of it; but consists generally of a fibro-adenomatous proliferation of the tissue of the gland, which immediately embraces the urethra, and is separated from the rest of the prostate by a layer of smooth muscle. The two lateral lobes are flattened by the proliferating mass and displaced to the sides. This explains why the hypertrophied tissue shells out so easily, and also that it does not form part of the lateral lobes, even when it appears to consist of two lobes. It also explains why the vasa deferentia are not interfered with, and why, fortunately, recurrences are so rare.

The wall of the bladder will already in the early stage present the appearance of a trabeculated bladder. In simple cases it is quite impossible to mistake the diagnosis.

We must take care not to confuse the early stage of tabes with enlargement of the prostate. This is quite possible if the tabetic patient micturates frequently and has residual urine. The trabeculated condition of the bladder would appear to support this mistaken diagnosis, unless one remembers that it also occurs in tabes. The distinguishing point is the fact that the tabetic has genuine incontinence. He allows his urine to pass long before his bladder is filled to its maximum, and therefore, as it were, runs away. We should at once be very suspicious if the patient is not well within the age when enlargement of the prostate is common, and if there is a history of syphilis.

Having diagnosed hypertrophy of the prostate, it becomes important to ascertain the *stage* in which the patient is, and also the *complications* which have taken place. The examination of the urine will show whether this is infected ; the use of the catheter immediately after spontaneous micturition will indicate whether the patient can empty his bladder completely or has residual urine ; and palpation of the kidneys will sometimes—by no means always—inform us whether any infection has ascended as far as the renal pelvis. The diagnosis of pyelitis can, however, be more securely based on lumbar pains, sometimes on the right and sometimes on the left side, on persistent digestive disturbances, and especially on acute attacks of retention, with fever, rigors, vomiting, diarrhœa, headache and occasionally slight delirium. These symptoms proclaim that the patient has arrived at the condition which Guyon has classically described as "urinaire," composed of signs of uræmia, at first intermittent and subsequently persistent, combined with septic absorption. Infection very often leads to the secondary formation of stones which are not necessarily free in the bladder, but which may be fixed in diverticula like a deposit in a boiler. They are most commonly found in the post-prostatic pouch, which so frequently forms in prostatic patients, and in which genuine diverticula may develop.

This **classical course** may be attended by several *variations*, which are important from the diagnostic standpoint. Sometimes the *symptoms appear to set in suddenly*. This may occur after indulgence in an abundance of liquor, when the alcohol temporarily paralyses the micturition mechanism, or when there has been more opportunity of filling the bladder than of emptying it; in these circumstances the patient wakes up to find that he cannot pass his urine. He has carelessly allowed his bladder to become overdistended, and the detrusor is no longer capable of overcoming the obstruction. On close questioning of the patient, one can generally elicit that he has had, of late, to get up frequently at night, and that the urinary stream has long lost the force which it possessed in his youth.

In other cases the first complaint does not concern difficulty in micturition, but *rectal tenesmus*, or some unpleasant sensation in the rectum or perinæum. One patient complained of feeling "as if he sat on a ball."

In some cases, *hæmorrhages* are predominant. They may be very profuse and cause rapid debility.

Hitherto we have been assuming that the prostate is found to be enlarged on rectal examination. But this is not always the case. A middle lobe may—not very often—be responsible for the urinary difficulty; or a hard prostate, although not very much enlarged, may be responsible, so that one may, with justice, speak of "prostatic patients who have no hypertrophy of the prostate." The cystoscope clears up all these points, and may also reveal a cancer which has been masquerading in the form of a slight hypertrophy.

The more often prostates are removed the more frequently does one come across carcinoma, instead of the expected innocent hypertrophy. A malignant neoplasm is at once suggested if we feel an uneven asymmetrical tumour in the prostatic region, growing towards the rectum, or if we feel a hard mass, which is not especially tender to pressure, but is sharply defined at the sides. If the cystoscope reveals an uneven irregular structure, instead of the two smooth swellings, we should make the same diagnosis. If a round circumscribed tumour develops in a completely asymmetrical manner the condition is very suspicious. The case is also suspicious if separate nodules can be felt per rectum in addition to what appears to be any ordinary enlargement of the prostate. The suspicion is strengthened if the patient begins to complain of sciatica, and if we find any metastases—especially in the skeleton, the diagnosis is established. It is sometimes possible to tell from its shape whether the tumour is carcinoma or sarcoma. If it is hard and uneven we think of cancer, if it is soft and roundish, of sarcoma. But, as already stated, operation has shown that cancer is often concealed within a hypertrophy which appears to be innocent, both to the examining finger and to the cystoscope. The suspicion of cancer is not confined to the cases of pronounced enlargement, but is shared by the small hard forms. It is not possible to be dogmatic, but we may say that every enlargement of the prostate, whose symptoms are on the increase, is suspicious of cancer.

The presence of cystitis or the formation of secondary stones do not help the diagnosis, because these may occur both with innocent hypertrophy or with cancer. But if persistent hæmorrhages occur the case is very suggestive of cancer.

(2) INFLAMMATORY PROCESSES.

Chronic irritative conditions of the prostate, such as occur in gonorrhœal cases, are of much less interest to the surgeon than **prostatic abscess** proper. If a patient suffers from rectal tenesmus and severe pain on defæcation, upon which symptoms there supervene shortly afterwards strangury, and possibly also complete obstruction of the urethra, it is obvious that there must be some acute inflammatory process between the rectum and the exit from the bladder, i.e., in the region of the prostate. If, on passing a finger into the rectum and on feeling over one of the lateral lobes, we detect a soft or elastic swelling, over which the mucous membrane is thickened like velvet, we diagnose an abscess. The speculum shows the mucous membrane to be œdematous and sodden, but otherwise it does not give such a realistic picture of the disease as the finger does. The introduction of a Nélaton catheter into the urethra will encounter a more or less definite obstruction.

Metal catheters should not be used in these cases, because they may easily injure the œdematous mucous membrane.

It sometimes happens that the whole clinical picture disappears suddenly by itself, with the discharge of pus into the rectum or into the bladder, or into both, a termination which disposes of the necessity to interfere.

The *cause* of the abscess is important from the point of view of prognosis. The principal question to decide concerns its gonorrhœal or tubercular origin, and there is not usually any difficulty in this. Gonorrhœa, even if not confessed to, may usually be recognized at the stage wherein it causes a prostatic abscess, by the remains of urethral discharge. But it is also necessary to be able to demonstrate the presence of the gonococcus. The discharge of the pus from the abscess into the urethra, which does not always pour out, but may only exude drop by drop, may completely resemble an active gonorrhœa, and it is therefore indispensable to examine the pus microscopically, when the history is negative.

A young man had a typical bilateral prostatic or periprostatic abscess. On examination, a few drops of thick pus exuded from his urethra, and he appeared to have had a recent gonorrhœa. But this suspicion could be put aside, absolutely definitely, for the bacteriological examination showed a pure culture of *Staphylococcus aureus*. The patient had recently suffered from a large boil on his sacrum.

The following case shows how serious prostatitis may be. A man, aged 52, suffered from a small boil on his face. This was followed by metastatic pneumonia and pleurisy. A prostatic abscess then supervened, and this led to thrombosis of the right pelvic veins and the iliac veins. Eventually there occurred an erosion (thrombotic ?) of the gastric mucous membrane and a fatal hæmatemesis.

If nothing points to gonorrhœa, we should think of tubercle, and if the symptoms have come on suddenly, the infection is a mixed one. A young man with a very tubercular family history became ill suddenly with typical symptoms of prostatic abscess. Gonorrhœa could be excluded. The abscess opened into the bladder, and the pus contained a pure culture of the *Bacillus coli.* A guinea-pig inoculated with the pus became tubercular. Nevertheless the focus in the prostate healed rapidly, so that without the inoculation the diagnosis would have remained doubtful.

CHAPTER LXXII.

INJURIES OF THE URETHRA.

THE injuries which concern the posterior portion of the urethra possess the greatest diagnostic interest. They are divisible into three groups: (1) Injuries from within the urethra; (2) injuries produced by external violence without causing a wound; and (3) the consequence of fracture of the pelvis.

False passages play an important part in the causation of **injuries from within the urethra.** A hæmorrhage, which is generally rather severe, warns the practitioner who passes a catheter carelessly, or the patient, that some damage has been done.

These injuries are most easily inflicted by the so-called English catheters, which were formerly in considerable vogue, and were given to patients for their own use as being harmless. Unfortunately they have not entirely gone out of fashion. They are not stiff enough for introduction without a stylet, but are quite stiff enough to do some damage.

Injury by such articles as pencils, nails, hairpins, &c., are less general. One should think of this possibility if a patient otherwise healthy—at any rate, physically—bleeds from the urethra, and has pain on micturition, apparently without cause. It would appear from English's summary that almost everything which could possibly get in has been found in the urethra.

Contusion of the urethra by violence which causes no external wound is of great practical importance. If one falls astride on the edge of a board, on a pommel, or a bicycle wheel or some similar object, the urethra is crushed between the pubic arch and surface on which it rests. A kick on the perinæum produces a similar result in a different way. The symptoms which are observed after such an injury indicate very distinctly the nature of the anatomical damage, even without passing a catheter. This proceeding, which is not always free from danger, should be the final step in diagnosis.

The following forms may be distinguished in this variety of injury:—

(1) If the patient has some trouble in micturating, but is able with a certain amount of force to empty his bladder of urine, which is free from blood, he has sustained a *peri-urethral hæmatoma* without any injury of the mucous membrane. The effusion of blood may be felt in the perinæum as a hard exudate. The catheter should not be used as long as the patient can pass his urine.

(2) If some blood comes with the first few drops of urine, although the bladder can be completely emptied, some slight *injury has been inflicted on the mucous membrane;* but the passage of a catheter is not justified unless signs of extravasation of urine appear.

(3) But there is another series of symptoms which occur with such constant uniformity that once seen, they can never fail to be recognized. The patient lies groaning, because, notwithstanding strong contractions of the bladder, and his own pressure, he can only evacuate pure blood from the urethra, although the bladder is quite full. There is a hard dark blue swelling which extends symmetrically like a butterfly's wings on either side of the perinæum. The longer one waits the more tense becomes the swelling and the patient's condition more agonizing. In the presence of such a clinical picture the diagnosis is clear enough without passing a catheter. The patient has a complete, or almost complete, rupture of the urethra, and all the urine which the bladder cannot retain is being extravasated into the connective tissue of the perinæum. The urine must inevitably decompose unless relief is given by operation. As a rule the catheter is held up at the site of injury, and it would only be a lucky accident if it entered the bladder by following up the anterior surface of the urethra, which sometimes is not torn through. The fact that such a lucky accident is possible justifies a cautious attempt at passing a catheter, but we must be careful not to draw a false conclusion. Clinical records often relate that the practitioner has withdrawn a certain amount of bloody urine, but the patient experiences no relief therefrom. If we make the attempt we shall arrive at the same result, and at the same time feel that we have not succeeded in entering the bladder. The explanation is probably that the urine, which is escaping from the bladder under pressure, has found a cavity for itself in the perinæum, which holds a certain quantity of blood and urine. In such cases we should not

persist with the catheter, but at once must provide for emptying the tissues of urine by a perinæal incision.

Obstruction of the urethra through a **fractured pelvis** is of less frequent occurrence. The considerations mentioned in connection with contusions from without, apply here also. The difficult evacuation of pure urine without blood strongly suggests compression of the urethra by a hæmatoma, such as would occur in the case of fracture of the symphysis. But the cause may be kinking of the urethral canal, due to its being dragged on by the displacement of the two pubic bones one against the other. If the normal function return within a few days, and if a catheter passed subsequently encounters no obstruction, the case is one of a simple hæmatoma. But if we still meet with some obstruction after the lapse of time—obstruction which cannot be overcome at all, or which requires some special form of catheter or some special manœuvre to do so—we must assume that there is a kink in the urethra due to displacement of the bones, although there may be little difficulty in spontaneously emptying the bladder.

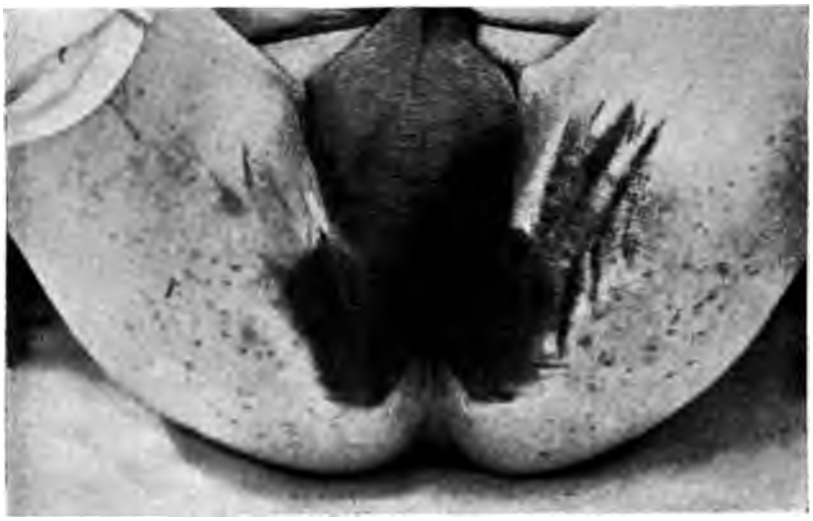


FIG. 259.—Rupture of the urethra through blow on perinæum. (Butterfly ecchymosis.)

In these cases the skiagrams show that one pubic bone overrides the other to the extent of one or more centimetres.

If blood is passed with the urine, the urethra is certainly injured—perforated, crushed, or lacerated by a fragment—but not torn right through. A catheter must not be passed in such a case, as long as the bladder can empty itself and there is no extravasation of urine.

If nothing but blood comes from the urethra, and the bladder fills up, while signs of extravasation of urine appear, we must decide to adopt the same treatment as in the cases which manifest the same symptoms after external contusion.

CHAPTER LXXIII.

SURGICAL DISEASES OF THE PENIS.

Injuries or **deformities** of the penis present no diagnostic difficulties. The so-called **fractures** or **dislocations** of the organ are only curiosities, and have little practical importance. **Constriction** is easy to recognize, if affected by means of the neck of a bottle or a female screw, but not so easy if a wire ring or a loop of thread has been employed. The last may cut so deeply into the penis that surgical interference is required to render it visible and to remove it. A similar condition is seen in **paraphimosis** caused by the retraction of a tight prepuce (fig. 260).

In the case of deformities, the first glance shows whether the cleft is above or below, whether **epispadias** or **hypospadias** is present. The degree of the latter is determined by the place at which the urethral orifice is situated.

Tumours and **ulcers** are much more important from the diagnostic standpoint. For practical purposes we must differentiate between subcutaneous growths on the one hand and inflammatory ulcers or ulcerated growths on the other hand.

(1) SUBCUTANEOUS GROWTHS.

These very rarely occur on the penis. **Sebaceous cysts** and **dermoids** are the only innocent growths which ever occur, and **sarcoma** the only malignant one. The former are either in the skin or under it ; the latter usually arises in the corpora cavernosa. The diagnosis presents no difficulty, but one must not mistake the hard nodular or cord-like induration which indicates **chronic inflammation of a corpus cavernosum** for a commencing sarcoma. **Bony growths in the penis,** which are very rare, are easily recognized by palpation and X-ray examination. **Elephantiasis**, a condition which is very frequent in the Tropics, is not really a growth, but it converts the penis into a club-shaped, and finally into an enormous and shapeless tumour. A similar condition occurs in other countries after repeated attacks of erysipelas, or after urinary fistulæ.

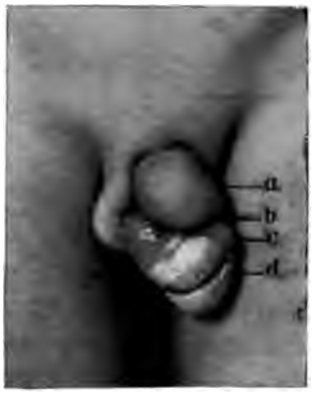


FIG. 260—Paraphimosis.

a = Root of penis. b = Constricting furrow. c = Œdematous and swollen prepuce, with internal layer outside. d = Œdematous glans penis.

Acuminate condylomata should also be mentioned here, although they are hardly of surgical interest. They are one of the very rare forms of tumour which are definitely infectious. As stated elsewhere, they have nothing to do with gonorrhœa, although they are often acquired at the same time as that disease.

(2) ULCERATIVE CHANGES.

In addition to venereal ulcers there exists a whole series of intermediate clinical forms, ranging from acute inflammatory conditions to an ulcerating cancer, which require complete exposure of the glans — sometimes with the aid of the knife—before they can be diagnosed.

(*a*) If there be pronounced inflammation of the foreskin and glans at an age when venereal infection is more or less infrequent, we should think of simple **balanitis** or **balano-posthitis.** In young lads, the cause is almost always phimosis; in old people it may also be due to retention of smegma or narrowing of the foreskin. If the inflammation is very pronounced or very obstinate, or if it dates from middle age, the urine should be examined for sugar—always assuming the presence of a certain degree of retention of smegma.

Sometimes considerable inflammation of the foreskin or glans occurs in the course of severe acute infective fevers.

It is, of course, obvious that the inguinal glands may enlarge in all these conditions. Extensive lymphangitis and phlegmonous complications may exceptionally occur.

(*b*) If venereal disease is not excluded—there is no age limit in regard to chancre, and even impotence is no guarantee against it—the prepuce and glans must be carefully examined, and a diagnosis of balanitis must not be made unless it is quite certain that no circumscribed ulcer is present. It is, of course, important not to confuse a superficial erosion with a genuine ulcer. Erosions may occur in any, case of balanitis, and are especially common in **herpes genitalis.** But they heal in a very few days with any mild treatment if the part is kept clean, whereas a real ulcer takes a long time to scar over.

Sometimes a hard chancre looks like a superficial erosion, but it can be easily distinguished from a harmless erosion by its indurated base.

(*c*) If the disease does not consist of any diffuse change, but merely of a circumscribed ulcer, we have to think of those conditions which we have already studied in connection with the oral mucous membrane, although, of course, they do not occur in the penis with anything like the same frequency. **A tubercular ulcer** has been observed on the foreskin and glans in cases of uro-genital tuberculosis, but it is extremely rare.

Instances have been recorded wherein tubercular lesions have followed suction of the blood by a tubercular operator, after ritual circumcision.

The problems of diagnosis mainly centre around the differentiation between soft and hard chancres, gumma, and cancer. An ulcer which appears a few days after sexual intercourse is usually a **soft chancre,** but it may become a hard chancre within two or three weeks. The former diagnosis is proved by the presence of Ducrey's bacillus and by the onset, within a few days, of diffuse infiltrating and painful buboes. The diagnosis of subsequent syphilitic transformation is proved by the obstinacy with which the chancre resists non-specific treatment, by the demonstration of spirochetæ, and by the serum test, but above all by the appearance of constitutional symptoms. If the ulcer has not appeared until after two or three weeks' incubation, the diagnosis of a **hard chancre** is quite certain; the discovery of spirochetæ and the onset of glandular enlargement after another couple of weeks, only serve to confirm the diagnosis.

The diagnosis is more difficult when a patient, who has just entered within the cancer period, denies all possibility of infection—an old proverb says "omnis syphiliticus mendax." No denial is of avail in the presence of a soft chancre with rapidly forming buboes, and the diagnosis of hard chancre only requires a little patience if a search cannot at once be made for the spirochæte. Consequently, there can only be any real doubt as between **gumma** and **cancer.** Experience shows that an error of diagnosis may damage a practitioner both in reputation and in money. If he amputates for a gumma, as has actually happened, he becomes responsible for wanton mutilation. If he treats a cancer for weeks or months as a gumma, he runs the danger of converting a curable malady into an incurable one, and the patient will make him responsible for the loss of the organ. But both mistakes are avoidable by a little examination. A *cauliflower-like cancer,* if definite, is immediately recognizable. This form usually follows an old phimosis, or may develop on a venereal scar, and in time ulcerates through the external foreskin. A *flat cancer* bears more resemblance to a syphilitic ulcer, but it lacks the fatty base of the gumma. The *medullary nodule-forming cancer* is quite unmistakable. According to Barney, Jews very rarely suffer from cancer of the penis.

All cancers which are covered over by the prepuce are characterized by a most offensive discharge. A microscopic examination, which will establish the diagnosis with accuracy before operation, should always be undertaken without delay.

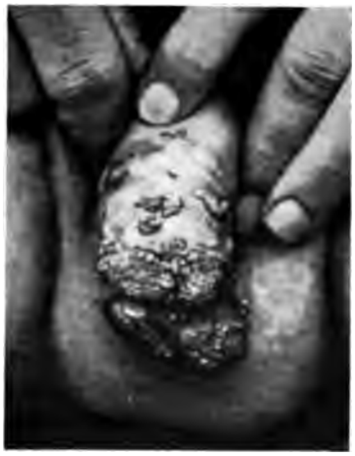


FIG. 261.—Acuminate condylomata.

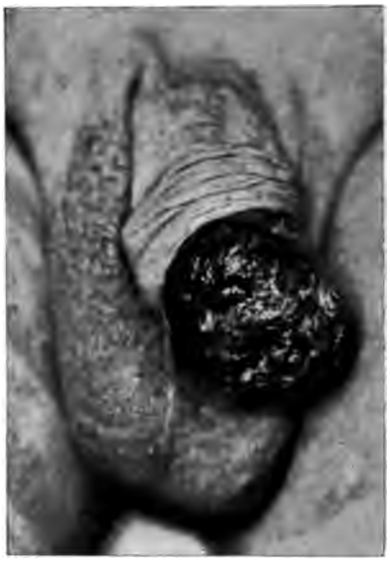


FIG. 262.—Cancer of glans penis.

We have already seen that the glands do not enlarge in cases of gumma, and that their enlargement *may* be absent in cases of cancer, or supervene at some later time, whereas the enlarged glands in connection with a hard chancre always appear at the classical moment.

Finally, some confusion is conceivable between a commencing papillary cancer and an **acuminate condyloma**, mentioned above.

PART V.

THE SURGICAL DISEASES OF THE PELVIS AND SPINAL COLUMN.

CHAPTER LXXIV.

TUMOURS OF THE PELVIS.

THE major portion of the pelvis is so extensively covered by soft parts, that it is very easy to overlook tumours in their early stage, even if they do not grow exclusively inwards. It is therefore most important to devote due and timely attention even to their indirect symptoms.

If a pelvic tumour grows inwards, it will sooner or later *press upon and displace the pelvic organs.* Bladder and rectal derangements will usually point to this result, and these symptoms demand a careful bi-manual examination, per rectum and the lower abdominal region.

The derangements which occur during labour from the presence of even small tumours within the pelvis are well known. The experienced obstetrician will always think, among other possibilities, of a new growth in the pelvis, when the head of a child refuses to engage normally.

Symptoms of displacement of the pelvic viscera are not always predominant in the clinical picture. If the growth extends mainly outwards, or if it is situated in the false pelvis, it gives rise to two other symptoms—the *appearance of a protuberance* in some part of the pelvis, and the result of *pressure on the nerve-roots.* A new growth of the pubic crest is distinguished by the early stage at which it can be seen and felt. It requires no further diagnostic consideration. But a growth more often announces itself by nerve disturbances long before it can either be seen or felt, and the patient is accordingly provided with various diagnoses and ordered to all possible spas.

A man, aged 52, had consulted many doctors for sciatica. At our first examination no organic cause for the neuralgia was evident. The rectum was free, the prostate normal, and nothing could be felt in the pelvis. The spinal column was also normal. The treatment for the sciatica had apparently secured temporary improvement. The patient returned in nine months' time, and then it was clear that the pains affected the region supplied by the anterior crural nerve rather than the sciatic nerve region; at the same time there was a striking weakness of the flexors of the thigh. The patient had to lift up his left leg with both hands to put it on the examination stool. In addition there were pains in the lumbar region; the twelfth dorsal vertebra and the centre of the sternum were painful to pressure. The diagnosis of "sciatica" was obviously discredited, and one had to think of organic damage to the great nerve-roots which supply the left leg. Meanwhile a tumour of the left iliac bone had become palpable on deep pressure, and this explained all the symptoms. A loud souffle could be heard with the stethoscope over it, and this confirmed the diagnosis of "sarcoma." The pain over the lumbar spine and the sternum indicated metastases, which therefore contra-indicated any operative interference.

It is not always pressure on nerves which suggests a concealed pelvic tumour; sometimes the pressure is exerted on *blood-vessels*, producing an increasing œdema of one leg. If both symptoms are present simultaneously, the suspicion becomes very great.

How can we tell whether a tumour which is found in the pelvis really originates from the pelvic bones? First, we must be able to exclude any connection between it and the pelvic viscera by considering the previous history and the actually existing symptoms. For instance, if a patient has for months been losing blood with his motions, and has symptoms of rectal stenosis with a growth adherent to the sacrum, he is not the subject of pelvic tumour, but of a rectal cancer which has contracted secondary adhesions to the sacrum. *Physical examination*, however, furnishes the clearest indications. Tumours which grow from the internal surface of the pelvic bones are usually more or less globular or uneven structures, connected with the pelvis itself at a narrowly circumscribed site. Malignant growths of the pelvic viscera, once they have become fixed to the pelvis, give the impression of a diffuse hard mass, which seems to have been poured out by the pelvic cavity. Innocent growths of the pelvic viscera never become so firmly fixed to the pelvis as to cause any mistake.

It might be quite possible to mistake a pelvic enchondroma for a firmly incarcerated fibromyoma; but examination under anæsthesia would clear this up, because some movement could then be obtained, even with a firmly incarcerated myoma.

In exceptional cases a very elastic, tense *burrowing abscess* which occupies the pelvic fossa, or the true pelvis, may be mistaken for a sarcoma, especially if it causes circulatory disturbances in one leg. But such an error would only be pardonable in the absence of any physical sign of tubercular disease of the spine or pelvic bones.

If a pelvic tumour has become large enough to be grasped by the hand, or to make special demands upon the tailor, there is no longer any difficulty in diagnosis. Tumours of a knotty structure within the pelvic cavity, especially in the vicinity of the ileo-sacral joint, are either **osteomata** or **chondromata**. They may, in the course of years, grow as large as a man's head, or even larger. They are not absolutely innocent, because they do sometimes produce metastases. But, in contrast to sarcomata, they do not cause any nerve disturbances until late. On the other hand, if we are dealing with a tumour which announced itself by pressure on nerves, before it could be demonstrated objectively, and whose symptoms are rapidly increasing, there can be no doubt about the diagnosis of "sarcoma." If auscultation shows that there is vascular engorgement (a systolic murmur), this tends to confirm the diagnosis of sarcoma.

Sarcoma of the acetabulum presents a special clinical picture. At first it suggests hip disease, but the early onset and persistence of neuralgia, combined with the absence of any disturbance in the mobility of the joint, makes the careful observer suspect something worse. Similarly, sarcoma of the ileosacral region is at first thought to be ileosacral tuberculosis. But it is just in this case that auscultation enables an early diagnosis to be made in some circumstances.

In exceptional cases the pelvic tumour is a secondary growth from a primary focus which remains undiscovered until death (prostatic cancer, hypernephroma, malignant glands). Sometimes pulsation can be detected over such secondary growths, and a systolic murmur may be heard, especially over a hypernephroma.

If the diagnosis still remains in doubt clinically, it can be settled by a *skiagram*, which will also show how much new bone formation there is in the tumour, the limits of its extent and the amount of bone destruction it may have caused.

In addition to the pelvic tumours hitherto discussed, one should mention the rare **fibromata** which grow from the iliac bone into the anterior abdominal wall. They usually occur in the female sex and their clinical behaviour approaches malignancy, so that they are very suggestive of the well-known fibromata of the abdominal integuments.

In considering the diagnosis of pelvic tumours one must think of **fibroma** and **sarcoma of the pelvic muscles**, which are most frequently found originating in the gluteal muscles. As long as they are movable while the muscles are relaxed, they present no difficulty in diagnosis, and their recognition either as a sarcoma or fibroma is made by their form and consistence and comparative rapidity of growth.

But this is not always possible, because some growths, which to the naked eye and microscopically appear to be fibromata, are liable to persistent recurrence, and their histological appearances may even change to those of sarcoma in the course of time.

If the tumour has become adherent to bone, even the skiagram may fail to indicate its site of origin.

The subject of pelvic tumours suggests reference to another class of tumours, which usually escape adequate discussion among abdominal tumours, because they do not originate in a viscus.

These are the **tumours of the connective tissue of the pelvis.** With few exceptions they are **dermoids,** which originate in the pelvic connective tissue, or rather in the perirectal tissue, develop above the levator ani, generally on the left side, behind the rectum. They are well known as causing obstruction in labour. If they grow chiefly upwards, they are usually looked upon as ovarian tumours adherent in Douglas' pouch—occurring as they mostly do in the female sex. If they extend in a downward direction, they are especially liable to be taken for burrowing abscesses. But their tense consistence, their well-defined roundish shape, and the extreme displacement of adjoining viscera should show that they are independent growths. Positive evidence is only furnished by exploratory puncture and operation.

The rule not to perform exploratory puncture until everything is ready for operation applies here just as well as on other occasions. Dermoids suppurate very easily and the portion of the body through which the puncture has to be made, does not always permit of thorough cleansing.

These dermoids are very rare in males, but when they do occur, and the patient is at the age of prostatic hypertrophy, they usually suggest this condition.

For eighteen years a patient of mine went about with this diagnosis. But after he had made a false passage for himself and even the village midwife failed to pass a catheter, he came to the hospital with a cyst the size of a man's head.

The same applies to the very rare **cysts of the prostate** or of the **retroprostatic connective tissue.** Sufferers from these tumours do not usually consult the surgeon until they get retention of the urine, and probably have a false passage in addition. The diagnosis is first made at the operation. In a few cases, hydatids have been found in this region.

CHAPTER LXXV.

CONGENITAL ABNORMALITIES IN THE SPINAL COLUMN.

IF a new-born infant presents a median swelling situated on the spinal column, either slightly or not at all movable, we should at once think of a "spina bifida." As it is not possible to distinguish all the finer differences in this malformation by clinical signs, we shall merely detail the main features upon which the diagnosis turns.

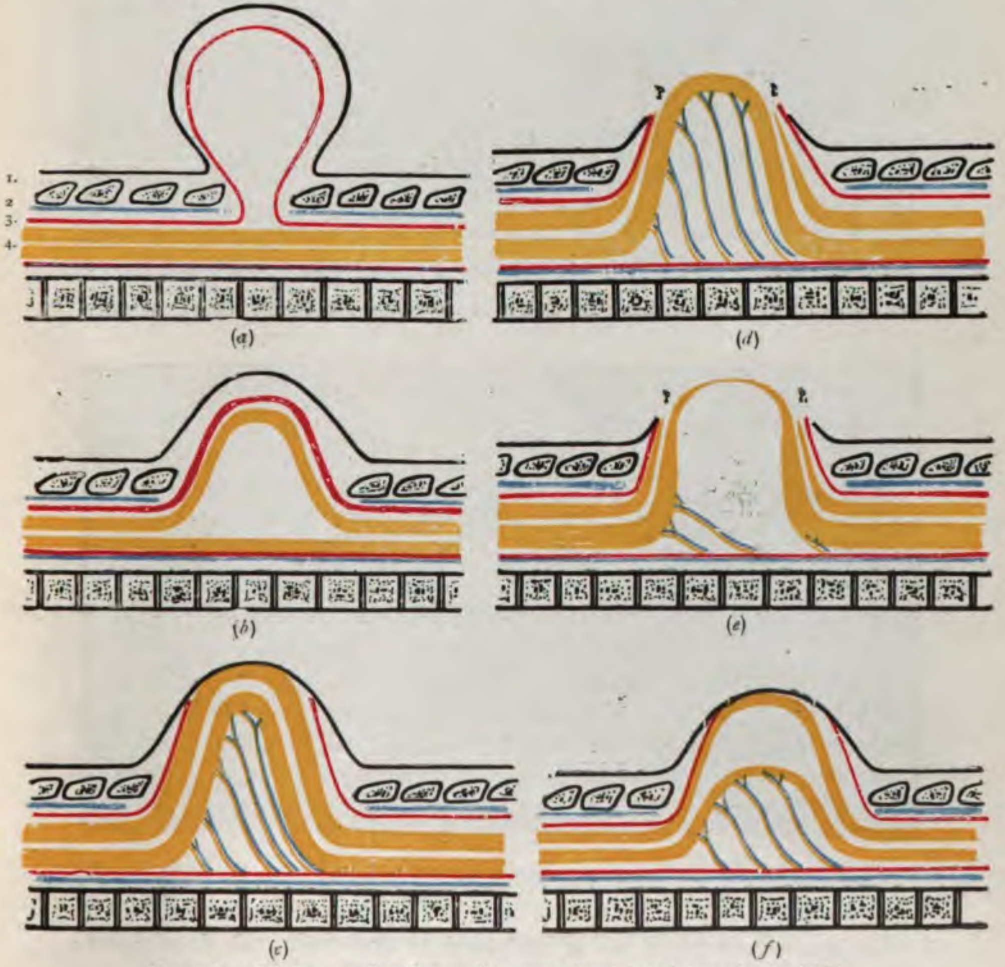


FIG. 263.—Diagrammatic scheme of the more important forms of spina bifida.

1. (black) Skin. 2. (blue) Dura. 3. (red) Pia. 4. (yellow) Spinal cord. P—P_1. Polar depressions. (*a*) Meningocele. (*b*) Myelocystocele. (*c*)—(*e*) Various degrees of myelo-meningocele. (*f*) Myelocysto-meningocele.

In some cases the spinal column, the spinal meninges and spinal cord are completely cleft, and form a hood over a shallow groove, or cause a proturberance, if a kyphosis is also present—**posterior rachischisis** (fig. 264). Other malformations, incompatible with life, usually exist at the same time, especially on the skull and in the brain.

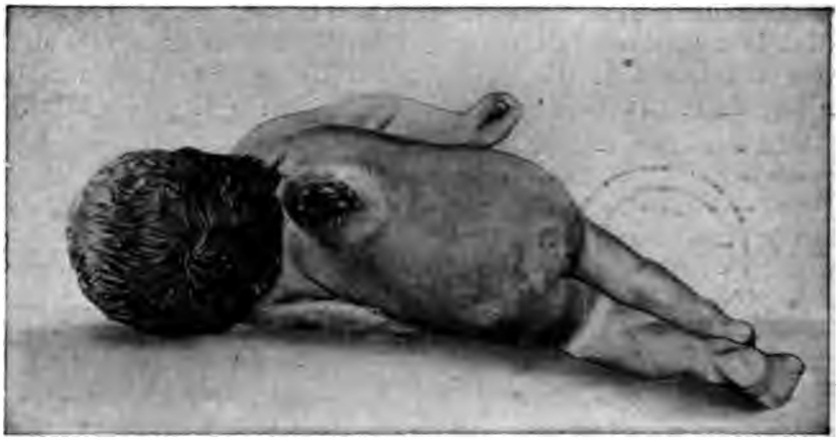


FIG. 264.—Posterior rachischisis. The dark portion corresponds to the medullo-vascular area.

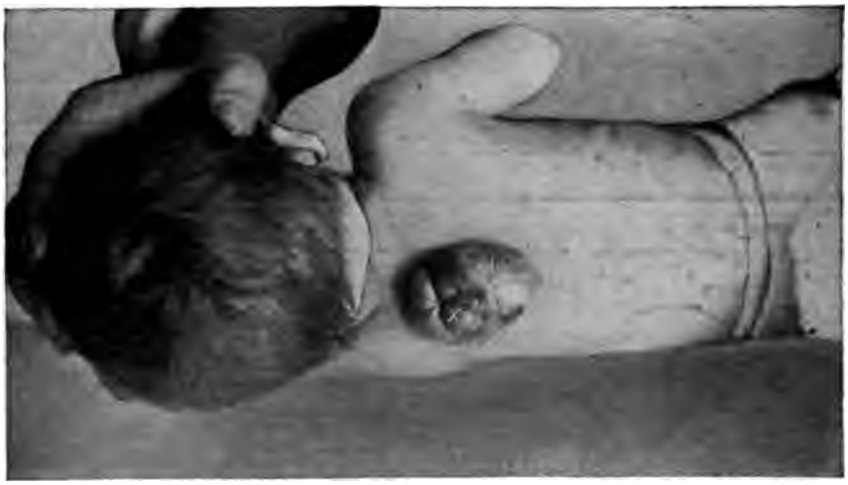


FIG. 265.—Myelo-meningocele with the superior depression clearly seen in the illus'ration (X).

In less severe cases the spinal cord is also cleft and gives rise to the reddish medullo-vascular area, but the extent of the fissure is less. The "tumour" is formed by an increased collection of cerebro-spinal fluid in the area of the soft spinal meninges on the ventral side of the hooded cord—**Myelo-meningocele** (fig. 265). This form merges by intermediate varieties into the form wherein the spinal cord is closed, but projects out of the canal, adherent to the posterior wall of the sac. The collection of fluid in this form is also found ventrally in the soft membranes, but is often found at the same time in the dilated central canal—**Hydromyelo-meningocele.** The tumour has normal skin at the periphery, and is covered at its top with fine scar-like epidermis (fig. 266).

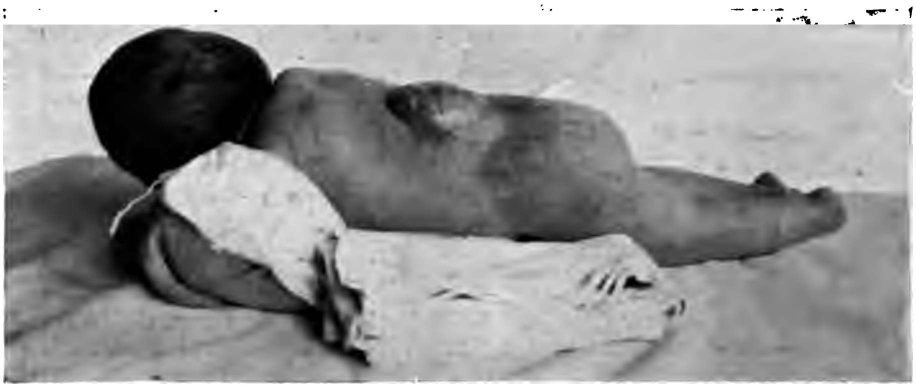


FIG. 266.—Myelo-meningocele.

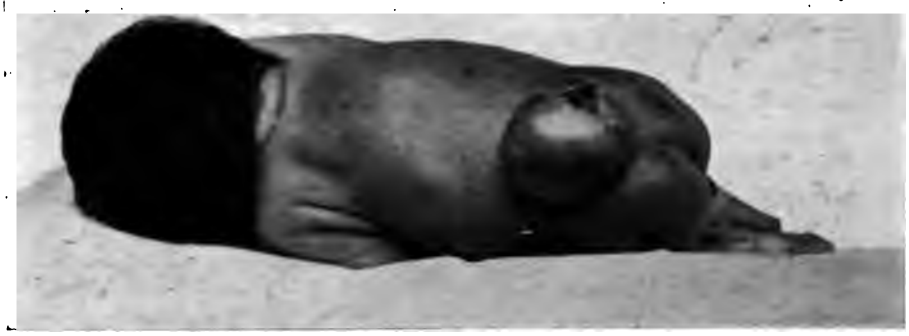


FIG. 267.—Myelo-meningocele in the region of the conus terminalis and the cauda equina.

If the spinal cord is free in the sac, and entirely covered by arachnoid membrane, and if the central canal is considerably dilated, the case is one of **myelo-cystocele.** If the tumour only consists of protruded arachnoid, the case is one of **pure meningocele.** The dura takes no part here either in the composition of the wall of the sac, but stops short at the level of the gap in the bone.

How can these various forms be distinguished clinically? In the first place by the rest of the child's condition. The more severe any other malformation, the more severe is the malformation of the spinal cord. Then the condition of the tumour itself is significant. If there be a medullo-vascular area present, or if a superior and inferior depresssion are found on an epidermal surface (fig. 265), one may be certain that the spinal cord is cleft. If tracts are seen through the sac wall, running from a dorsal thickening thereof towards the spinal column, they will be recognized as nerve-roots, and we may assume that the spinal cord, whether cleft or not, is adherent to the dorsal wall of the sac. If such tracts are absent, the case is either a myelocystocele or a meningocele. But the latter may also contain in its wall coiled-up nerve-roots, running back to the spinal canal, and thus the diagnosis may be rendered difficult. When the sac is very large the diagnosis can be facilitated by examination through transmitted light, and also by the circumstance that a pure meningocele only occurs in the sacral region.

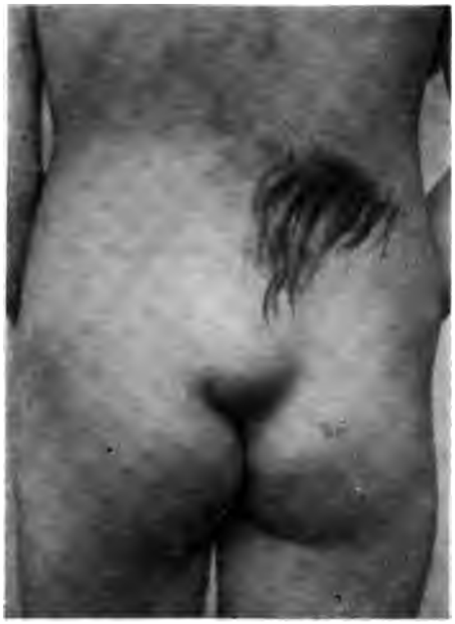


FIG. 268.—Meningocele with tuft of hair.

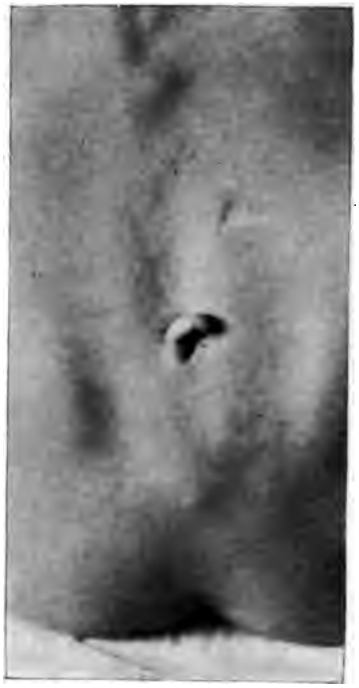


FIG. 269.—Vestigial tail.

Special diagnostic interest attaches to those somewhat infrequent cases wherein the cleft formation of the spinal column and the change in the spinal cord or in its cavity are so indefinite that they are not observed on a superficial examination. The patient, however, seeks advice about slight sensory or paralytic symptoms, or occasionally about trophic disturbances of the lower extremities. Sometimes the disturbance is one of muscular balance, leading to talipes equinus or cavus. If the symptoms are specially of a motor character, one is inclined to attribute them to poliomyelitis. But, on an inspection of the back, we will at once be struck by the well developed hairiness of an area, usually situated in the lumbar region. The hairs are generally arranged in a semicircle, transversely to the spinal column, with its convexity downwards. They often become quite long. Slight cicatrical changes are frequently seen on the skin. On palpating the spinal column a gap will be noted in the series of vertebral spines, at the level of the hairy patch. In this gap there is an elastic swelling, which usually varies in size from a pea to a cherry. This constitutes the malformation known as **spina bifida occulta.** The swelling, which is felt, may either be a *pure meningocele* or a *myelo-meningocele.* In some cases there is not even any nervous disturbance, and the only indication of the malformation is the abnormal hairiness.

When assistant to Kocher, I saw a peasant lad in whom temporary paralytic symptoms of the lower limbs supervened after a blow on the back. The abnormal hairiness of the lumbar region easily led to the diagnosis of spina bifida occulta, which had been injured by the blow.

Finally, the sacral region is the site of all possible congenital tumours: Fibromata, angiomata, lipomata, sarcomata, teratomata. The last include an unbroken series from simple dermoid cysts to fœtal implantations and double monsters. These tumours are sometimes isolated, at others they are associated with clefts in the spinal column and spinal cord. Sometimes the peculiar character of the tumour allows it to be identified as a fibroma, lipoma, dermoid or lymphangioma ; but as a rule one must be content with the diagnosis of **congenital sacro-lumbar tumour,** and further detail must be left to the microscope. If a dermoid ruptures it gives rise to a *dermoid sinus*, already referred to among the perinæal fistulæ.

If the sacrum has an appendage looking like a tail, it can very rapidly be decided by palpation and by a skiagram whether the structure only consists of soft tissue—a *false tail* (lipoma, fibroma) or is a vestigial tail without bone — or whether it contains a prolongation of the spinal column and constitutes a *real tail.*

These vestiges of a "tail" period are never found among an entire people, as has been stated. They occur in individuals (generally males) of all races, and vary from a modest stump to a quite conspicuous structure, even resembling the tail of a pig.

CHAPTER LXXVI.

LUMBAGO.

MANY a diagnosis, made with a pretence of learning, is merely a classical term used as a cloak for our ignorance. This is very often true of "lumbago." A pain in the lumbar region, whose cause we do not know, is usually provided with this name. We cannot apparently dispense with this term, but we should reserve it for a pain which comes on suddenly. It is because of the suddenness of the pain and its unknown causation that it is called in German "Hexenschusz" (witch's shot). This excludes a large number of lumbar pains which have nothing to do with general lumbago. Among these may be mentioned as the more important the lumbar pains of tabes and paralysis; spinal caries, renal stone and tuberculosis; chronic colitis (especially on the left side). The lumbar pains which occur in acute febrile diseases, from influenza to small-pox, are also excluded. Our discussion of the subject is, therefore, limited to the two conceptions—**rheumatic lumbago** and **traumatic lumbago.** Formerly, the malady was called either rheumatic or traumatic, according to personal taste, and the nomenclature had no further significance.

But since insurance against accidents has become the vogue, there is hardly any condition as fertile as lumbago as a cause of actions for damages. The blame for this is not only to be attributed to the proverbial greed of the insured for compensation, but to some extent to the circumstance that the diagnosis of rheumatic lumbago is based on very insecure foundations. Here, as elsewhere, a condition which we cannot explain is termed rheumatic.

No one who has ever suffered from lumbago, and, therefore, observed its progress with accuracy, can possibly believe that a genuine inflammation, be it of rheumatic or other nature, can set in with such suddenness. It always originates in some slight and unexpected movement which the spinal column is not prepared to meet by the fixation of its joints. The first consequence of this want of fixation is that the lateral articulation gives way, and the final result is twisting of this joint. There can be no doubt that some people suffer from an effect of this kind more than others; but this is no evidence for the inflammatory origin of the pain. This does not, however, dispose of the contingency that pains may also arise owing to rheumatic inflammation of the muscles of the back or of the lumbar nerves. But although these pains apparently begin without any direct cause, they are not sufficiently sudden in their onset to merit the designation of lumbago. The confusion between the two conditions is due to the fact that the pains in both are similarly localized, and are of the same subjective character.

Thus we see that there is no sharp line of demarcation between the ordinary and the traumatic form of lumbago ; the transition between them is gradual. The actual strain which causes traumatic lumbago may be so slight as to be well within the range of normal movements ; the traumatic effect is simply due to the neglect of the individual to fix the spine in anticipation of the movement.

As far as the legal definition of "accident" is concerned, the term "traumatic lumbago" should be limited to injuries which result from abnormal movements of the spine, such as over-bending, over-straining and excessive twisting—movements which are calculated to produce distortion, rupture of muscles, and tearing off of articular and transverse processes, despite any amount of muscular fixation.

In both groups of cases the most striking feature consists of the muscular rigidity of the affected spinal segment, generally, but not always, the lumbar spine. There is, in addition, a certain amount of localized pain on pressure and on movement; but no sign is absolutely conclusive in the differential diagnosis. A definite diagnosis can be more satisfactorily based upon a correct knowledge of the trauma on the one hand, and the course of the symptoms on the other hand.

Reliable information as to the injury is often unattainable, because the patient's statements are apt to be exaggerated. It is only when there has been some evident cause (lifting an unusually heavy weight, a fall, or external violence) for a severe trauma, that we should regard the symptoms which are present as due to an "accident" in the legal sense.

The course of the symptoms furnishes conclusive evidence of the diagnosis, except in the case of insured patients, who have an interest in the prolonged duration of their malady. Ordinary lumbago disappears in a few days, but the consequences of an extensive rupture of muscle, of a severe twist, or the tearing off of bone, may persist for weeks and months. The diagnosis is easy enough if a good skiagram reveals a torn fragment of bone (fracture of a transverse process). But in the absence of this, or of evidence supplied by a hæmorrhagic discoloration of the skin, a few days after the accident, pointing to a superficial hæmatoma, we are bound to rely on the *bona fides* of the patient, and on careful observation in hospital.

The differential diagnosis receives little aid from inquiries directed to previous rheumatic symptoms, because genuine lumbago has nothing to do with rheumatism.

It is much easier to recognize pain in the back which follows a *direct contusion*. The nature of the trauma is obvious, and we often find its immediate consequences in abrasions and ecchymosis of the back.

Compression fracture of the spinal column can only be mistaken for lumbago if the history has not been taken into account, and the symptoms of compression-fracture are unknown to the practitioner.

It is important to realize that fragments of bone may be broken off by indirect violence—either through muscular contraction, or the dragging of ligaments. Such small fragments may be missed, even on careful X-ray examination, especially in fat patients.

Cases which show no physical symptoms, but wherein the subjective complaints are severe, constitute the most difficult of medico-legal problems. The patients delay their return to work from time to time, extending over a period of years, whereas they would resume their occupation in a few weeks or months if they were not insured. The worst cases are those which are "credited" with a fracture, on the strength of one of the many anatomical variations in the vertebral column.

CHAPTER LXXVII.

INJURIES OF THE SPINAL COLUMN.

INJURIES of the spinal column resemble those of the skull, in that their study is dominated by the associated injury sustained by its contents. In our examination, chief attention must be devoted to this consideration.

Let us assume the case of a patient who has had an injury to the spine, generally through a fall from a height, or through something having fallen on him, and who then complains of his back. But he comes on foot, and presents no change in the shape of the spine, nor any symptoms connected with the nervous system. He is suffering either from a contusion or distortion, or possibly from a fracture of a spinal or transverse process. In the worst circumstances, he has sustained a compression-fracture. It is only in the case of injuries in the region of the cervical spine that victims can walk about when the damage is serious, e.g., dislocation. If the injured person has to be carried on a stretcher, and he lies thereon unable to stir, he has either lost the supporting power of the spinal column, or has sustained an injury to the nervous system, or possibly may be suffering from both simultaneously.

We only propose to deal here with the more serious injuries of the spinal column, and will start with a concrete case.

When the patient came into hospital his posture was as depicted in fig. 270. On the following day, when the circulatory disturbance had increased, he presented the posture shown in fig. 271, and the hyperæsthetic zone was displaced one segment higher up. In a few days there was so much improvement that even the eighth segment resumed its functions; but death from broncho-pneumonia followed three weeks later. Fig. 273 illustrates the spinal injury which had occurred in this case.

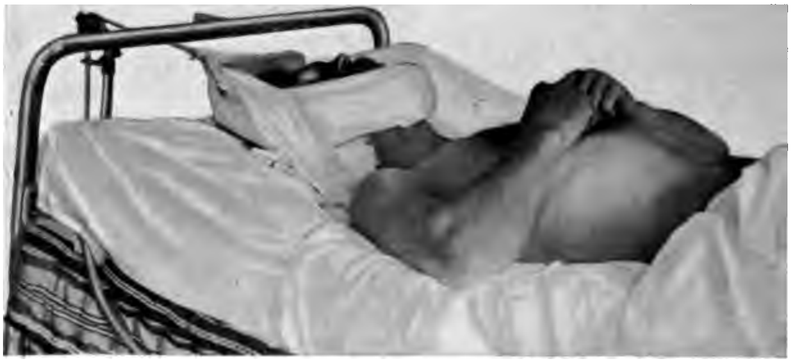


FIG. 270.—Posture of arms in a transverse lesion at the level of the seventh cervical segment.

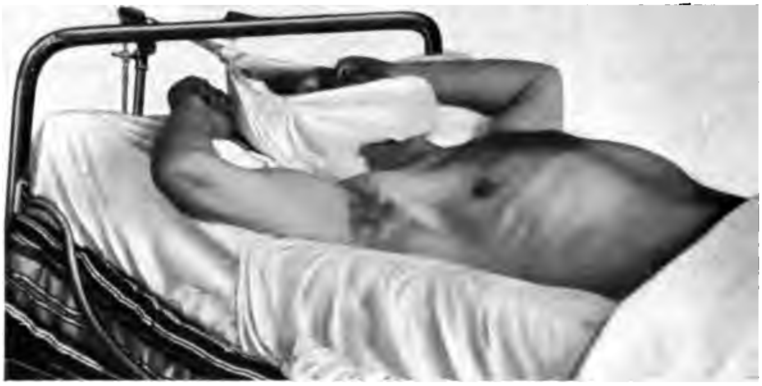


FIG. 271.—Posture of arms in a transverse lesion at the level of the sixth cervical segment.

(2) After this summary examination of mobility we proceed to test **sensation.** If this is preserved in the lower extremities, although possibly weakened, or lost to certain stimuli, a complete lesion can be excluded, even if motion is entirely absent. But if it is entirely lost, as is usually the case when there is total motor paralysis of the corre-

incontinence has once started, is especially found in cases of partial damage to the spinal cord. We know that this may also occur in dogs after a complete lesion, but the subject has not been sufficiently worked out in man.

On examining the rectum, it will be found to be full of fæces, as long as they are solid—retentio alvi. If the rectal contents are liquid, they escape involuntarily—incontinence of fæces.

Extreme meteorism, which is a sign of *intestinal paralysis*, is another symptom of visceral derangement. It is a sign which has often suggested genuine intestinal obstruction, and which has led to the performance of laparotomy.

We must finally refer to disturbances in the *innervation of the pupils*. If reflex fixation of the pupil be present with miosis, it follows that the pupillo-dilator fibres are interrupted somewhere in their course through the spinal cord, and that therefore the lesion must be above the first dorsal segment, in the roots of which these fibres leave the cord.

(5) There now remains the important matter of the examination of the **skin and tendon reflexes.**

In a complete lesion the **superficial or skin reflexes** are usually lost, but they return again. Kocher has, however, pointed out that the genital reflexes are not lost, viz., the erection reflex, and unilateral contraction of the lower abdominal muscles on squeezing the testicle (Kocher's testicular reflex). The **tendon reflexes,** especially the *patellar reflex*, are of great importance. If it is absent, it signifies a very severe injury, generally a complete lesion. If it remains permanently absent there is no doubt at all about this (Bastian-Brun's law). It may also be absent in partial lesions. It does not then usually take more than a few hours for it to return, though I have seen the absence persist for days in rare cases. If, on the other hand, the patellar reflex remains after an injury to the spinal cord, or is actually increased, we may safely exclude a complete lesion, even if the other symptoms are severe.

These remarks only apply to human beings and to sudden traumatic complete laceration. In *dogs*, the tendon reflexes may also, in such a case, return after a short delay. In *man*, the reflexes are either retained or even increased, in cases of *gradual* interference with the cord, through tumours or inflammatory processes. On the other hand, there is no record of a case wherein the tendon reflexes had previously been normal, and in which they were either retained or increased after the spinal cord has been suddenly torn through. I will not dispute the possibility of a partial return of the tendon reflexes months after a complete traumatic division of the cord, even in man, but I have never seen such a case; but this does not invalidate the diagnostic importance of Bastian-Brun's law.

II.—DIAGNOSIS OF THE NATURE, DEGREE AND POSITION OF THE INJURY.

Our examination now puts us in a position to answer the two important questions to which every spinal injury gives rise, namely :—

(1) Is the injury complete or partial ?

(2) At which level is it situated, and what conclusions may we draw from the injury to the cord, in regard to the injury to the spinal column ?

(A) THE DEGREE AND THE NATURE OF THE SPINAL CORD INJURY.

We have first to decide whether the injury has caused *complete* or *partial* division of the cord. We may summarize the indications already referred to in the following way :—

We may assume a complete lesion when there is persistent, symmetrical, total, flaccid motor paralysis, with sensory paralysis in the corresponding area, and when the tendon reflexes are lost for a considerable time, and when the bladder and rectum are paralysed, in the absence, however, of all motor and sensory irritative symptoms in the paralysed regions.

We must, on the other hand, assume a partial lesion when signs of voluntary innervation and of sensation are present below the site of injury ; when in their absence, the patellar reflex is retained or is soon restored ; when motor or sensory symptoms of irritation appear in the paralysed regions, in the first few days after the accident, and when bladder and rectum still act voluntarily, or at any rate when their automatic function sets in early.

A partial lesion can naturally occur in various degrees and forms.

Hemisection of the cord produces a somewhat typical form of lesion with a symptom-complex to which the name of Brown-Sequard's paralysis has been given. The more complete and the sharper the hemisection, the more accurately do the symptoms correspond to the following scheme, viz. :—

On the *side of the injury* :—

(*a*) Motor-paralysis, in the form of paralysis of a pyramidal tract, with a localized zone of cornual paralysis at the upper border.

(*b*) Vasomotor paralysis.

(*c*) Hyperæsthesia for superficial sensibility (tactile).

(*d*) Loss of deep sensibility (muscle sense).

(*e*) In the cervical cord ; paralysis of the oculo-pupillary fibres.

On the *uninjured side* we find :—

Sensory paralysis for pain and temperature ; sometimes diminution of tactile sensibility.

It would be of great interest from the point of view of treatment, if we could go further and distinguish between *contusion* and *compression*. The present state of our knowledge, however, does not allow us to do so.

We might very well indirectly conclude that contusion had taken place if we can demonstrate displacement of a vertebra. If we have concluded, from the absence of this sign and from the slightness of the symptoms, that the case is one of mere compression by a *hæmatoma* (always a very uncertain diagnosis), then the presence of much blood in the cerebrospinal fluid, drawn off by lumbar puncture, would suggest that the hæmatoma is situated inside the dura. This intradural hæmorrhage is also distinguished from hæmorrhage inside the cord (hæmatomyelia) by the predominance of *symptoms of irritation*, (paræsthesia, increase of the muscle tone and of the reflexes.)

In **hæmatomyelia** the *paralytic symptoms* predominate ; if the hæmorrhage is in the cervical cord, the disturbance is most marked in the lower extremities. The sensations of pain and of temperature undergo most disturbance, just as in syringomyelia. Very circumscribed hæmorrhages lead to **diplegia**, and as they are most frequent in the cervical cord, the diplegia is of the brachial type. Finally, it should be noted that such hæmorrhages in the cord (especially in the grey substance) may occur without any injury to the spinal column, as they have been observed to follow a temporary *overstrain* of the spine and consequent *dragging on the cord*. These have always been situated in the cervical or in the lumbar cord.

(B) THE POSITION OF THE SPINAL CORD INJURY.

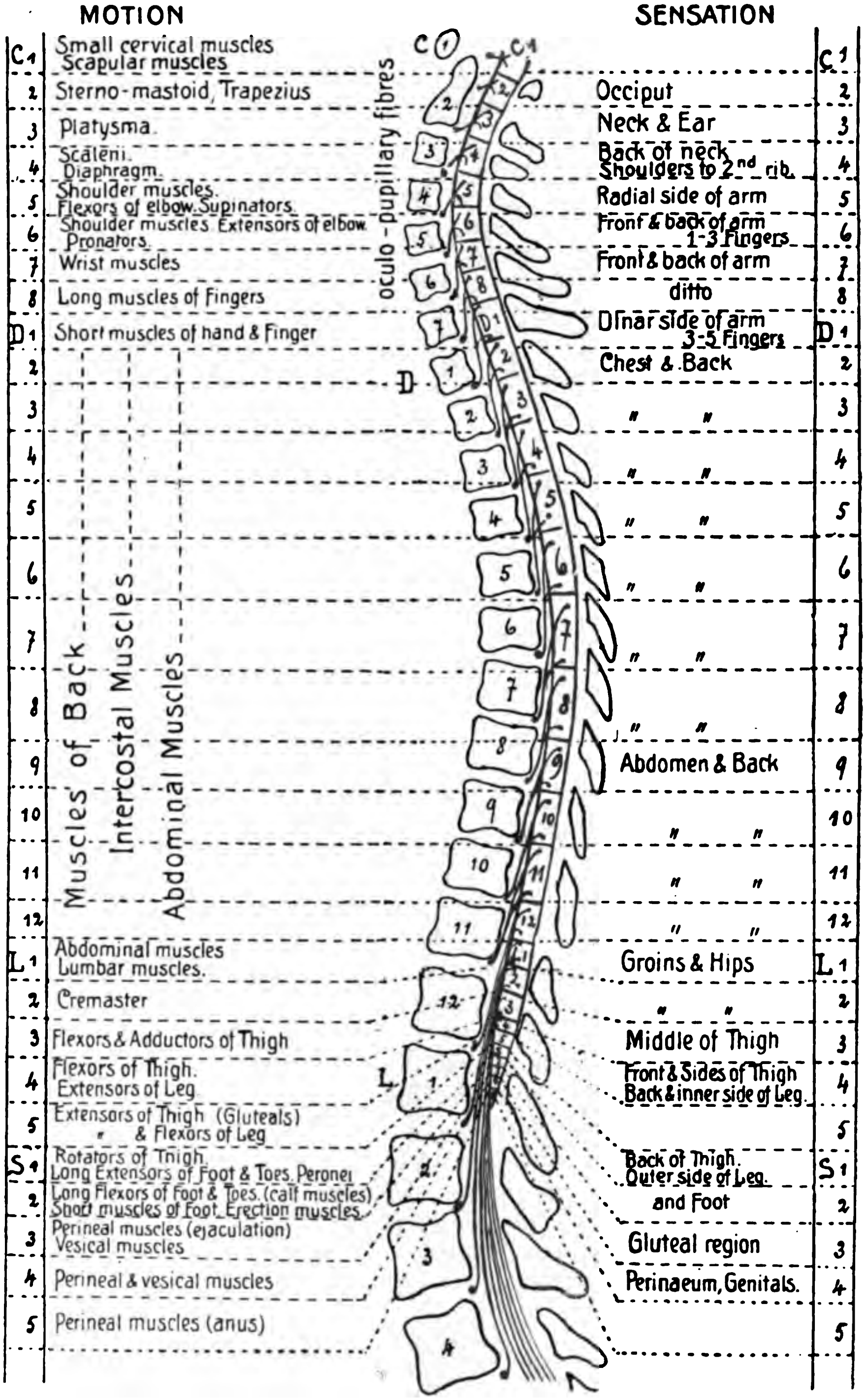
(The Diagnosis of the Level of the Lesion.)

The level of the lesion may be ascertained by co-ordinating the results of the tests for motion and sensation. We need not enter into details, because they are clear, from Plates I, II, and III, which are diagrammatic representations of the distribution of motion and sensation in accordance with the individual segments, and also from the adjoining diagram (fig. 272). It is only necessary here to add a few general remarks.

(1) In injuries of the cervical cord the posture of the arms tells us at once the approximate level of the injury, as previously stated. We must, however, not be content with the demonstration of these postures, but must carefully examine the *power of movement*.

In comparing what we find with the plates, we must remember that motor symptoms are not all due to the same cause. They may be caused :—

(*a*) By compression, or the tearing through of the pyramidal tracts.



	MOTION	SENSATION
C 1	Small cervical muscles Scapular muscles	
2	Sterno-mastoid, Trapezius	Occiput
3	Platysma.	Neck & Ear
4	Scaleni. Diaphragm.	Back of neck Shoulders to 2nd rib.
5	Shoulder muscles. Flexors of elbow. Supinators.	Radial side of arm
6	Shoulder muscles. Extensors of elbow. Pronators.	Front & back of arm 1-3 Fingers
7	Wrist muscles	Front & back of arm
8	Long muscles of Fingers	ditto
D 1	Short muscles of hand & Finger	Ulnar side of arm 3-5 Fingers
2		Chest & Back
3		" "
4		" "
5		" "
6		" "
7		" "
8		" "
9		Abdomen & Back
10		" "
11		" "
12		" "
L 1	Abdominal muscles Lumbar muscles.	Groins & Hips
2	Cremaster	" "
3	Flexors & Adductors of Thigh	Middle of Thigh
4	Flexors of Thigh. Extensors of Leg	Front & Sides of Thigh Back & inner side of Leg.
5	Extensors of Thigh (Gluteals) " & Flexors of Leg	
S 1	Rotators of Thigh. Long Extensors of Foot & Toes. Peronei	Back of Thigh. Outer side of Leg.
2	Long Flexors of Foot & Toes. (calf muscles) Short muscles of Foot. Erection muscles.	and Foot
3	Perineal muscles (ejaculation) Vesical muscles	Gluteal region
4	Perineal & vesical muscles	Perinaeum, Genitals.
5	Perineal muscles (anus)	

FIG. 272.

(*b*) By destruction of the anterior cornua and of the intramedullary roots.

(*c*) By damage to the roots after their exit from the spinal cord or from the dural sheath.

According to earlier views of spinal cord pathology, the first form should cause spastic paralysis, without the reaction of degeneration, the other two should cause flaccid paralysis with the reaction of degeneration. But, as a matter of fact, all paralyses are flaccid at first, and even when due to a break in the pyramidal tract, the spasticity does not come on for some considerable time, indeed until the lower spinal segment has regained its automatism. But, on the other hand, the reaction of degeneration enables us to distinguish between paralysis due to a break in the pyramidal tract and paralysis due to a lesion in the cornua or in the roots, at any rate after a few days. The nuclei are, however, not acutely divisible into segments, so that the nerve supply of one muscle may be derived from several segments. The reaction of degeneration, however, only manifests itself definitely when the whole of the nucleus or all the roots are destroyed. If a transverse lesion for example, hits off the upper end of a nucleus, only a small part of the muscle undergoes trophic change. The rest of the muscle is paralysed owing to the break in the pyramidal tract, and we must not expect the reaction of degeneration. Patients with severe damage to the cervical cord have generally succumbed before the reaction of degeneration has had time to develop.

We are apt to diagnose the lesion in too low a segment, owing to the fact that auxilliary innervation may be derived from the segment next highest to the one affected. On the other hand, the upper limit of the symptoms may, in exceptional cases, be due to an ascending *traumatic myelitis*, or to some transitory distant cause, and so we may diagnose the lesion higher up than it really is. It is, therefore, of the greatest importance to make repeated examinations, comparing the one with the other before deciding as to the level of the injury.

In general, we may say that the shoulders and the elbows are supplied from the fifth and sixth segments, the wrist from the seventh, the long muscles of the fingers from the eighth, the small muscles of the hand and fingers from the first dorsal segment, which is functionally connected with the eighth cervical.

We examine *sensation* on the arms, because the neck and shoulder, as well as the thorax as high as the level of the second rib, are supplied by the fourth segment (supraclavicular nerves). The radial side of the arm corresponds to the fifth cervical segment, the ulnar side to the first dorsal segment. The areas corresponding to the other segments lie between them in the form of bands. As the segments also coalesce in regard to sensation, we must only make use of complete loss thereof, for the purpose of focal diagnoses, at any rate in total lesions.

In Plate III, the left side represents the areas of spinal root innervation, and the right side represents the areas of cutaneous nerve distribution, according to Villiger. In regard to the former, we have followed Edinger, and have not depicted sharply defined areas, but have graduated the tone of the colours to indicate the overlapping of innervation. When the lesion is partial we define its level by the zone in which the disturbance is most pronounced and in which the most forms of sensation are lost, that is to say, we do not take into consideration the sensibility to touch only, but also to pain and temperature.

Symptoms of irritation are of more importance here than in the case of motor disturbances. They do not, in cases of a total lesion, indicate the actual segment which is injured, but the one directly above it, sometimes even one higher. This is the case if auxiliary fibres are irritated in the area corresponding to the next higher segment. In partial lesions of the cord, the irritative symptoms may also correspond to the injured segment itself. We may assume this to be the case, when irritative and paralytic symptoms are combined in the same zone, and when there is no complete paralysis below it.

Ascending myelitis may affect sensation just as it does motion, and therefore give rise to the diagnosis of a segment, which is one too high.

(2) In lesions of the *cord in the dorsal region*, the *motor* conditions are not of great value for focal diagnosis, because neither the nerve supply of the muscles of the back, nor that of the intercostals can be applied to the purpose. The nerve supply to the abdominal muscles, derived from the seventh to twelfth dorsal segments, are equally useless for this purpose.

The *sensory* nerve supply is, therefore, of more importance. As previously remarked, the supraclavicular nerves, derived from the fourth cervical segment, supply sensation as far as the second intercostal space. Next to this lies the area of the second dorsal segment, with its border transverse, and not parallel to the ribs, and the other dorsal segments follow. These areas lie lower than the point of exit of the corresponding nerves in the upper part of the chest to the extent of three spinous processes, and lower down to the extent of four or five.

Kocher has suggested a very convenient indication, according to which the upper border of insensibility corresponds to the lowest anterior point of the intercostal space in which the injured nerve runs. From this point the border line runs, not obliquely, but rather horizontally backwards. The areas widen out in the neighbourhood of the linea alba, because the area of the twelfth intercostal nerve reaches as low down as the symphysis.

What has been said of the cervical cord, in regard to the limitation of the areas, applies here also.

The behaviour of the *pupils* will show whether the first or the second dorsal segment has been destroyed, as previously stated.

(3) As the segments of the *lumbo-sacral cord* are very small in extent, we may expect several to be involved in one injury. The cornual lesion predominates over the paralysis of the pyramidal tract, and the return of automatic activity to the cord below the site of injury is compromised by the great extent to which it is injured. The shortest reflex arcs are very liable to be directly interrupted, so that some reflexes do not return, even after prolonged delay. The diagnosis is also rendered difficult owing to the course of the nerve trunks being more oblique than in the other parts of the spinal cord, and therefore more subject to extensive contusion. It may even be difficult, under these circumstances, to decide whether there is a genuine lesion of the cord, or merely a contusion of the cauda equina.

In actual practice, the following may be taken as a guide: if, in a case of complete motor and sensory paralysis, some reflexes are still obtainable, the condition is certainly one of *cord lesion;* a comparison of the physical features found on examination with the appropriate plates, will show the *precise position* of the lesion. If no reflexes are obtainable, we may be in doubt, especially for the first day or two, whether the cord or the cauda equina has been injured. In such a case the onset of irritative symptoms, "paraplegia dolorosa," would point to *compression of the cauda equina.* If later on, some reflexes return, despite the persistence of the paralysis, it shows that the cord has, at any rate, participated in the injury. If the reflexes are persistently absent, the onset of the reaction of degeneration in all the paralyzed muscles, strongly suggests contusion of the cauda equina, whereas the persistence of electrical irritability in some of the paralyzed muscles would indicate that the cord is also injured. Subsidence of the paralysis and a simultaneous return of the reflexes naturally also occurs in contusion of the cauda equina. The *position of the injury in the spinal column* is obviously of significance. If it can be shown that it is situated above the first lumbar vertebra, it is conclusive of injury to the cord; if the injured vertebra is much lower down, it is equally conclusive of injury to the cauda equina. It is important to distinguish between an injury to the spinal cord and to the cauda equina, because the prognosis of the latter is much better. Relief of the compression by operation, even after a year or two may result in recovery, even from complete paralysis.

A young man, who was hurt in a motor-car accident, sustained, among other injuries, a contusion in the sacral region. He exhibited for a few weeks nervous disturbances, which, from his description, appeared to be due to a lesion of the cauda equina. Examination, however, showed that the only trace of his accident consisted of great increase of the tendon reflexes, especially on one side. This was conclusive of injury to the cord.

Many *types of paralysis* have been described in connection with the lumbo-sacral cord, as for the cervical cord—almost as many types as segments. It is quite unnecessary to enumerate them, because they can all be inferred by co-relating the motor and sensory nerve areas as depicted in the plates. They are not so striking to the observer as the types of cervical cord injury, recognized by the peculiar postures of the extremities.

(C) RELATIONS BETWEEN THE INJURY TO THE CORD AND THE VERTEBRÆ.

Just as clinical examination and skiagraphy often enable us to diagnose the level of a lesion in the cord, so, on the other hand, are we able, in some cases, to diagnose the displaced vertebra by the segment affected. A few anatomical data are necessary for this.

We begin with the *cervical vertebræ*. The cervical cord has eight segments, and as the first dorsal segment lies behind the last cervical vertebra, it follows that the seven cervical vertebræ correspond to nine segments. The segment in the middle of the cervical cord must be one higher in number than the corresponding vertebra. At the end of the cervical vertebræ, the number of the segment is one and a half to two higher; thus behind the 6th vertebra, we have, not the 6th segment, but the 7th and a portion of the 8th, and behind the 7th vertebra there are the remainder of the 8th and the whole of the 1st dorsal segment.

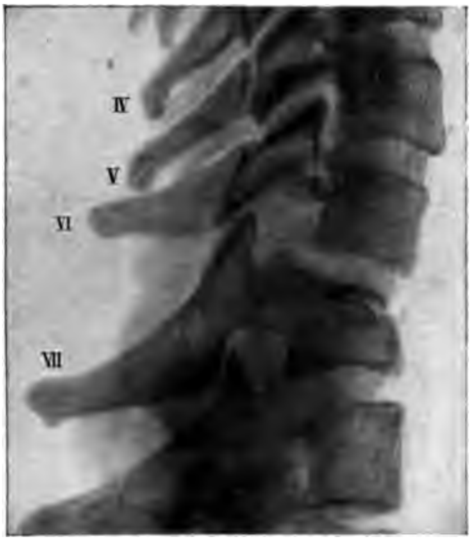


Fig. 273.—Dislocation-compression-fracture in the dislocated position. (From a *post-mortem* preparation). The 7th cervical vertebra is compressed, and the 6th is displaced slightly forwards. The 5th vertebral spine overrides the 6th. The articular processes of the 6th and 7th are not interlocked, but their extremities are in contact with each other.

In the *dorsal vertebræ*, eleven segments (2-12) are divided between the first ten vertebræ. In the upper of these vertebræ the number of the segment is one higher than that of the corresponding vertebra, whereas in the lower vertebræ the difference is two. Thus the 3rd segment lies behind the 2nd dorsal vertebra, while the 12th segment is mainly behind the 10th vertebra.

The whole of the *lumbar and sacral segments* lie behind the 11th and 12th dorsal and the 1st lumbar vertebræ. It is not practicable to separate these segments anatomically. We may, however, say that the upper edge of the 12th dorsal vertebra corresponds to the 2nd lumbar segment, and that the upper edge of the 1st lumbar vertebra corresponds to the 5th lumbar segment.

If we apply on the basis of these statements and the table in fig. 272, the existing nerve disturbances to the *indirect diagnosis of the injured vertebra,* we must remember that the cord is not contused by the anteriorly displaced vertebra, but by the upper edge of the one immediately below (fig. 274). Sometimes the cord is contused by a small piece of the upper displaced vertebra, resting on the posterior edge of the vertebra below (fig. 277). In cases of compression-fracture the cord may be damaged by a fragment of the vertebral body, forced into the spinal canal.

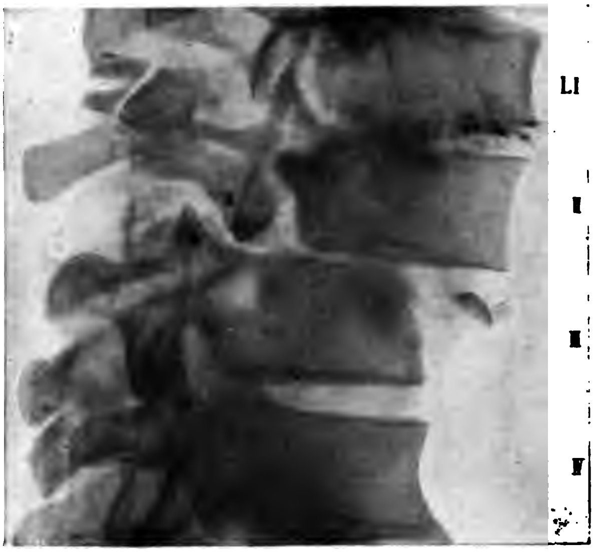


FIG. 274.—Complete bilateral dislocation between the 2nd and 3rd lumbar vertebræ. Interlocking of the articular processes a and b. Indication of an oblique fracture at the anterior border of the 3rd lumbar vertebra. (From a *post-mortem* preparation.)

If we wish to verify the diagnosis based upon the nerve symptom, and obtain a *direct determination* of the displaced or injured vertebra we must be careful to move the patient with the very greatest caution.

Anticipating what will be said later, we will observe here only that the spine of a vertebra displaced forwards, either by dislocation or by fracture-dislocation, is depressed, and generally turned somewhat upwards, and that it, therefore, limits anteriorly the gap found in the row of spinous processes. If, on the other hand, one individual vertebra is compressed, its spine projects somewhat backwards as the summit of a more or less pronounced angular kink in the spinal column. If several vertebræ are compressed, their spines will form a round curvature.

(D) THE FORM OF THE SPINAL INJURY.

It is of therapeutic and prognostic importance to recognize the form of the spinal injury. If this is not elucidated by a skiagram, we must depend upon the indirect evidence furnished by the spinal cord injury, or by the signs found in the spinal column.

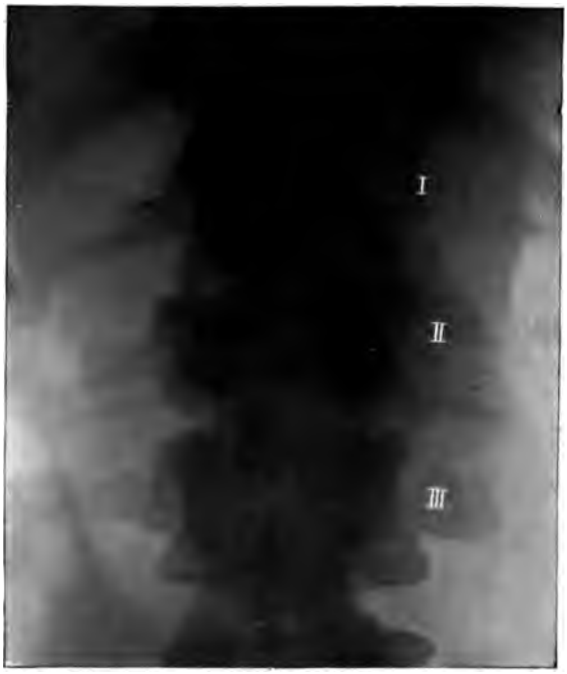


FIG. 275.—Compression-fracture of 2nd lumbar vertebra. The vertebra lower than the adjoining ones. Intervertebral disc narrower than normal.

We may distinguish, in accordance with old custom, between *dislocations and fractures,* at any rate, on paper. The *dislocations* are either unilateral (rotation-dislocation), or bilateral (total dislocation). They are incomplete when the articular processes override each other (fig. 273) and complete when the articular processes are interlocked (fig. 274). *Fractures* concern either the arch and processes only, or the body itself. The latter, which claim our main interest here, are either compression-fractures (figs. 275, 278, 279), or oblique fractures, i.e., fractures which traverse the body of the vertebra obliquely, from above and behind to below and forwards. These oblique fractures sometimes involve two neighbouring vertebræ. It is only rarely that the line of an oblique fracture ascends from one side to the other. There are finally some fractures, intermediate between oblique fractures and compression fractures, in that the wedge-shaped fragments exhibit signs of shattering by pressure, in addition to their wedge-like form. The right to distinguish between oblique and compression fractures follows from the different ways in which the two injuries occur. In compression-fracture the force acts in the axis of the spinal column; in oblique fractures it acts more or less vertically to it. The more these two forces co-operate the more mixed is the type of the fracture. The more the force acts perpendicularly to the spinal column, the more displacement takes place between the two vertebræ, and the final effect of the injury is to resemble a dislocation. If the displacement has produced complete dislocation we describe

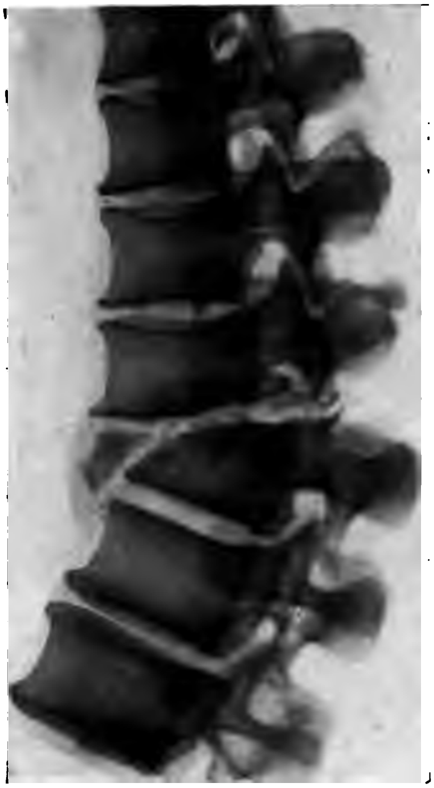


FIG. 276.—Oblique fracture-dislocation. (From a *post-mortem* preparation.)

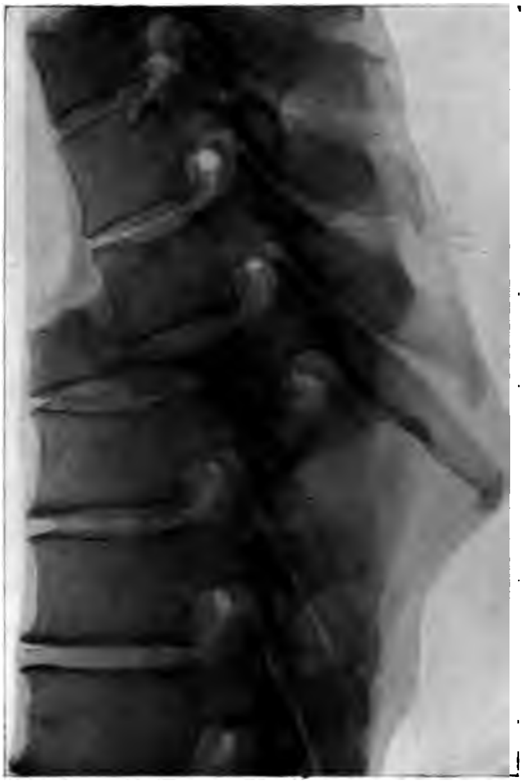


FIG. 277.—Compression-fracture-dislocation, with displacement of upper section, backwards. (*Post-mortem* preparation.)

it as a *total dislocation fracture*, which may be either an *oblique fracture dislocation*, or a *compression-fracture dislocation*, according to the degree and form of the shattering.

How much can we recognize clinically of all this? The main question which concerns the future of the patient is this: Is he suffering from an injury without displacement and therefore generally without severe contusion of the cord, or from an injury with displacement and therefore with more or less severe contusion thereof? Compression-fractures and fractures of the arch belong to the first group, total dislocations and total dislocation-fractures in their various forms belong to the second group—injuries which we may class together as *total displacements*.

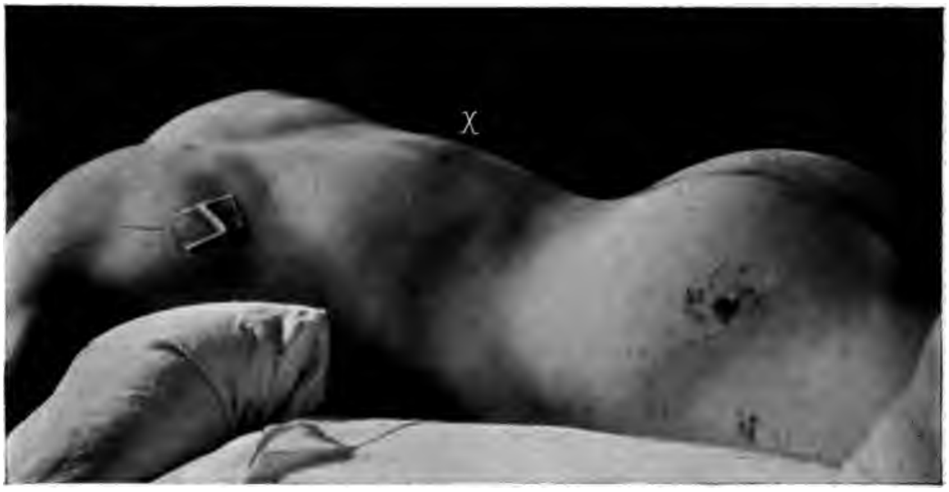


Fig. 278.—Compression-fracture of the lower dorsal vertebræ (X). Very slight cord symptoms. Simultaneous localized compression-fracture of 3rd dorsal vertebra and transverse fracture of the sternum.

(1) Fractures of the Spinous and Transverse Processes.

Fracture of a spinous process is caused by a direct localized force, and the objective signs consist of a circumscribed persistent pain on pressure over the spinous process, striking preternatural mobility thereof, and the subsequent onset of ecchymosis. As a rule, however, a positive diagnosis can only be made from a skiagram, taken from the side.

It is more difficult to recognize fracture of a *transverse process*. This injury may result from direct violence or from muscular action. It may be suspected from the presence of a persistent and pronounced pain on one side, on lateral flexion of the spinal column, and from pain on pressure on one side, while the corresponding spinous process is not painful. A positive diagnosis can only be made after an X-ray examination. This injury is practically confined to the lumbar vertebræ, and often causes prolonged discomfort. The actual pain may not be very great, but it suffices to incapacitate people from employment for many months, especially when there is no ardent zeal for work.

Fracture of a transverse process in the neck is directly combined with fracture of the *articular process.* The mechanism of rotation dislocation usually comes into play, but the interlocking of the articular process is prevented by the fracture of one of them, so that the position of rotation is not fully developed. Under these circumstances the diagnosis is very difficult. The symptoms are too severe for a simple sprain, and insufficiently distinct for a unilateral dislocation. The differential diagnosis is only possible by X-ray examination.

We thus see that opportunities for wrong diagnoses are very abundant, unless X-ray examination is employed, especially when the patients are workmen insured against accidents. Formerly, if the symptoms did not coincide with the physical signs, some observers called the patient a malingerer, others of a more kindly disposition called him the subject of a "traumatic neurosis." But it has become evident that pure malingering is rare, and that the term traumatic neurosis ought not to be applied unless the accident has produced serious results, accompanied by physical damage. The view now taken of the cases where there is no anatomical injury, is that it is neither a matter of malingering nor of traumatic neurosis, but rather of—often unconscious—exaggeration.

Several skiagrams are often required before the case becomes clear. It is important not to mistake for fractures some of the frequent anomalies which occur in the lumbar spinal and transverse processes.

(2) Fracture of the Vertebral Arch.

If there are no spinal cord symptoms, there is every probability that the arch has been fractured. If, however, cord symptoms are present we should think of fracture of an arch, if the spinal column has not lost its supporting power, and if axial pressure is only slightly, or not at all painful, and when these symptoms are combined with a severe pain on pressure over one spinous process, possibly with some anterior displacement thereof, and moreover if there is also a local hæmatoma and the injury has been direct and narrowly circumscribed.

The early diagnosis of fracture of the arch is important from the point of view of treatment. It is the only form of fracture in which, when complicated by damage to the cord, early operation is clearly indicated—the elevation or removal of the depressed arch—and in which very good results are obtained. Fissures sometimes exist in the vertebral arches, especially in the lumbar region. These must not be mistaken for fractures.

(3) Compression-fracture.

This is suggested when the cord symptoms are slight, or quite absent, when the spinal column loses its posture either incompletely or not at all, but when—in contrast to fracture of the arch—there is very pronounced pain on axial pressure. Caution is required in applying this test. The injury is usually in the nature of a blow in the long axis of the body, being produced by a fall from a height on the head or the feet, or on the buttocks. The bodies of the vertebræ, which are chiefly composed of spongy bone, yield more readily to these blows than the arches and the articular processes, which are mainly composed of compact bone. The crushing of *one* vertebral body causes the spine to bend forwards in an angular manner, but when *several* vertebral bodies are involved, as is usually the case, the bending is more like a kyphosis. When only one vertebra is compressed, the curvature may be limited to a slight prominence of the spinous process of the affected vertebra, and a hardly perceptible angular kink in the spinal column, whereas if several vertebræ are crushed there is always a round curvature (fig. 278). Sometimes one has to search carefully for the symptoms, for the fracture may easily be overlooked in the absence of cord symptoms, especially if our attention is diverted to other injuries besides those of the spine, or if the patient walks about again soon after the accident. The persistent heavy pain would then be the only indication.

These compression-fractures are most frequently found in the dorsal and lumbar vertebræ. When situated in the upper dorsal vertebræ, our attention is often directed thereto by a *transverse fracture in the upper portion of the sternum.*

Slight compression-fracture in the lumbar vertebræ does not usually cause any visible kink or protuberance. The change of form is just sufficient to straighten out the normal lordosis of the lumbar spine.

It frequently happens that vertebræ which have been damaged by a compression-fracture undergo secondary absorption of bone, and then give way after long delay, so that a protuberance appears months after the accident—possibly also with nervous symptoms. This is known as Kümmel's disease, and is also, inappropriately, termed traumatic spondylitis. (See Chap. 78.)

A skiagram of a compression-fracture, taken in an antero-posterior view, shows that the level of the body is depressed, sometimes with structural changes, and nearly always narrowing of one or both contiguous intervertebral discs. If there be any doubt, a skiagram with a profile view should be taken. This shows the wedge-shaped vertebral body and the decrease in size of the intervertebral discs.

Axial contusion of the spinal column represents the slightest degree of damage by a blow in its long axis, the intervertebral discs being especially damaged. We may diagnose this condition when, after such an injury, there is pain on axial pressure, but neither any change in shape nor cord lesion.

(4) Complete Dislocation.

Complete dislocation may be diagnosed when a severe or complete cord lesion exists, and when the spinal column has completely lost its posture. The latter symptom is not always present in complete dislocation in the cervical spine, because the ligaments may retain some supporting power. The cord lesion is much less severe in these cases than in fracture-dislocation.

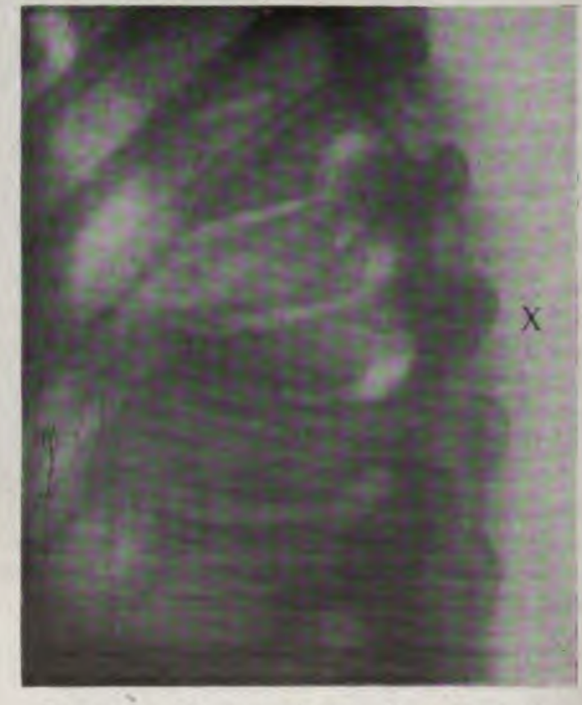


Fig. 279. — Compression-fracture of dorsal vertebra. Lateral view, taken during life. (8th dorsal vertebra wedge-shaped, intervertebral discs narrowed.)

A very powerful trauma is required to cause a complete displacement, and it must, at any rate partially, be of the character of an over-bending (very rarely over-stretching). The diagnosis must be based, as in compression-fractures, on the presence of change in shape of the spinal column, apart from the chief symptoms previously mentioned. But the nature of the injury is such that the dislocation may rectify itself spontaneously by appropriate posture, and on examination it may not be possible to discover anything beyond a spinous process which is painful on pressure and is depressed. The absence of any striking change in form does not, therefore, exclude a complete displacement as long as indirect symptoms thereof exist. In the cases wherein the change in shape persists, we find an increased interval between two spinous processes in the usual flexion fractures. The spinous process, which limits the gap above, is depressed forwards, in consequence of the displacement of the vertebral body, with which it is connected. If two vertebræ are broken, this gap is found between the spines of these two vertebræ, and here also the lower spine is the more prominent.

It would be too much to expect any further details in diagnosis, and to decide between the various forms of complete displacement, in the living patient without a skiagram. Indeed, the matter has no practical significance. It is more important to be able to distinguish between complete dislocation and dislocation-fracture in the cervical spine, because the former is capable of being correctly reduced, and the reduction should be carried out. Complete dislocation without interlocking of the articular processes, is in no way distinguishable from dislocation-fracture, because in the latter case also, the kinking and displacement may be rectified by appropriate posture (spontaneous reduction). It is quite different, however, if complete dislocation is combined with interlocking. Whether the head is bent forwards or backwards it is always anteriorly displaced in relation to the back, and

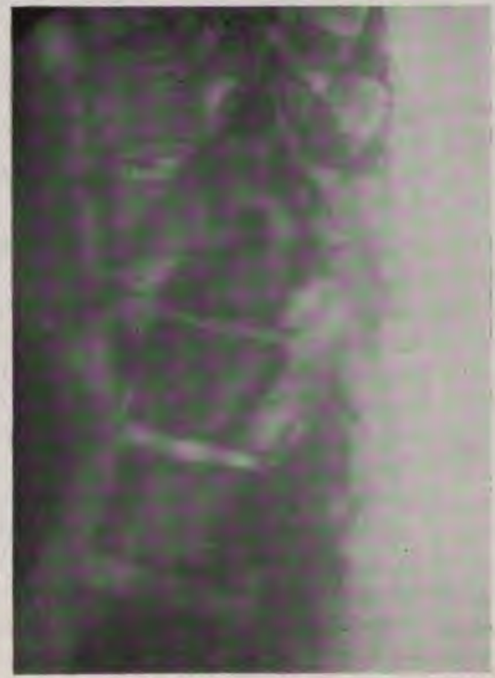


FIG. 280.—Dislocation-fracture (oblique). Lateral view, taken during life. Spinal canal considerably obstructed.

there is no tendency for it to return to its normal position, either spontaneously or by appropriate posture. This immovability and the freedom of the cord should always suggest a complete dislocation rather than a dislocation-fracture, and we should therefore attempt reduction. A skiagram furnishes conclusive evidence.

Complete dislocations, pure and simple, have been observed in the upper dorsal vertebræ, but they resemble dislocation-fractures in every respect, and it is quite impossible to effect any reduction, as in the case of dislocations in the neck.

CHAPTER LXXVIII.

THE SURGERY OF NON-TRAUMATIC DISEASES OF THE SPINAL CORD.

THE recently qualified practitioner during the first few years of practice usually endeavours to classify the spinal cord diseases which he sees under one of the schemes he learnt as a student. But the more their memory begins to fade and the more he realizes his therapeutic helplessness, the simpler become his diagnoses, and he finally limits himself to such groups as tabes, syphilis, paralysis due to spinal caries, infantile paralysis, and "obscure diseases of the spinal cord." The patient suffers no great harm from this process of simplification, unless his case happens to be one which surgery can cure, or, at any rate, relieve. This applies especially to tumours within the spinal canal.

Not all the tumours which damage the spinal cord possess equal interest for us. If an obstinate sciatica or an intercostal neuralgia comes on a few years after an apparently successful operation of cancer of the breast, it is easy to diagnose a secondary growth in a vertebra; but, unfortunately, the therapeutic interest is *nil*. If a period of unexplained neuralgia is followed by the appearance of a tumour on the surface of the spinal column, and if the spine kinks simultaneously with the sudden onset of a paraplegia, there is no difficulty in diagnosing a primary malignant growth, but the therapeutic significance is no greater than in the case of a secondary growth. But, in addition to these very frequent incidents, tumours which are accessible to operative treatment occasionally occur. Such are the innocent tumours of the *spinal column*, which grow into the spinal canal, especially *osteomata*, *fibromata*, and *chondromata* and *hydatid cysts*, and also *new growths* and *inflammatory granulation tumours* of the *spinal cord* and its coverings.

Root symptoms, mostly of a *sensory character*, appear first as a rule : i.e., localized unilateral neuralgias, hyperæsthesia, and, finally, anæsthesia or, at any rate, hypo-æsthesia. *Motor* symptoms very rarely appear first. It is only in cases of tumour of the cauda equina that the root symptoms are from the first symmetrical and both sensory and motor in character.

It is obvious that these root symptoms do not exist, or at least are not pronounced, if the position of the tumour does not encroach upon the roots.

Pressure upon the spinal cord itself begins after a certain time, varying with the growth of the tumour. The result of this pressure is more or less loss of the conducting power of the cord. If the tumour has a *lateral* situation the symptoms are essentially those of *Brown-Sequard's hemiplegia.*

Otherwise, the only difficulty in determining the exact relation of the tumour to the spinal cord or the extent to which it has penetrated, is due to the fact that not only must one take into consideration the anatomical position of the growth but also the susceptibility of the individual nerve-tracts to pressure.

The following questions present themselves as the suspicion of a spinal cord tumour arises :—

(1) Is a Tumour Actually Present?

(*a*) We may begin with the stage of *root symptoms*. One should think first of tabes in the differential diagnosis from the common spinal cord diseases; but this condition is easily recognized by its characteristic symptoms, especially by the loss of the knee-jerks. In tumour of the cord these are increased. Then it is important to decide whether the symptoms may not be due to an early **spinal caries** or a rare **hypertrophic pachymeningitis**; but the symptoms are usually bilateral in these conditions, whereas, in cases of tumour, they are always unilateral at first. They are also much less severe in pachymeningitis than in tumour. It is more difficult to exclude a commencing *spinal caries;* indeed, it is quite impossible to make a differentiation as long as the symptoms are unilateral and no change in the spine can be detected either clinically or by X-rays, and the patient himself is not tubercular nor possesses hereditary disposition thereto. The greater frequency of *herpes zoster* in cases of tumour furnishes only a very indefinite indication, and nothing but the continued progress of the case can elucidate it. In spinal caries the root symptoms either become bilateral in the course of a few months or the cord symptoms subside, but in cases of innocent tumour the root symptoms may remain unilateral for years without any other essential changes occurring.

The appearance of spinal column symptoms, viz., pain on pressure, prominence of a spinous process and pain on axial pressure, disposes of the possibility of spinal cord disease and pachymeningitis, as well as of tumour of the spinal canal. The diagnosis must then lie between **spinal caries** and **sarcoma of the spinal column**; but the differentiation is sometimes impossible. The fact of the much greater frequency of spinal caries, however, justifies the practitioner in diagnosing that condition. If the case is not eventually cleared up by the appearance of a burrowing abscess on one side or the other, or by metastatic growths, then we may follow Bérard in deciding upon the possibility of a tumour if confinement to bed and weight extension produce no improvement. A skiagram should also be taken, because the rapid onset of extensive destruction indicates sarcoma. The neuralgia associated with sarcoma is more intense than that of caries.

(*b*) If the patient has *spastic paraplegia* without any deformity in the spinal column we should not, even then, entirely exclude spinal caries, although this would be a very rare event. The case is more likely to be **chronic myelitis,** or **multiple sclerosis,** and if the lesion is in the cervical cord, **hypertrophic pachymeningitis.** If a neuralgic stage preceded the paraplegia, or if radiating pains persist, we may exclude myelitis and multiple sclerosis. If the paraplegia was not preceded by pain, the diagnosis may be in doubt, or exploratory operation may be indicated, for cases of spinal cord tumour occur wherein sensory root symptoms are completely absent. On the other hand patients often describe the paræsthesis of multiple sclerosis as "pain."

(*c*) If *root and cord symptoms have occurred simultaneously with deformity in the spinal column* the diagnosis lies exclusively between **spinal caries** and **malignant growth.** If a burrowing abscess be present, it decides the matter, but sometimes a **hydatid cyst** which has reached the surface has been mistaken for such an abscess. In the absence of all objective indications, we must rely upon the history, and if the symptoms have existed for years we must attribute them to spinal caries; if their course has been rapid and does not extend beyond months, they must be attributed to a sarcoma. Very vascular sarcomata are sometimes recognizable by loud murmurs.

(2) What is the Nature of the Tumour?

The remarks already made in connection with tumours of the brain apply also to **solid tuberculomata** and **gummata** of the cord. The history should guide us, but not control us.

Solitary tubercles may exist in the spinal cord, exhibiting all the symptoms of tumours. They may be shelled out like tumours—an operation which has been attended by good results.

In neighbourhoods where the **echinococcus** is endemic, we should think of the possibility of hydatids, and our diagnosis would be confirmed if the patient presented any other localization of this disease. Symptoms which have persisted for years, without causing any appreciable change in the spinal column, suggest a more or less **innocent tumour of the spinal canal,** especially of the **spinal meninges.** Schlesinger holds that a tumour which has persisted for more than three years is generally intradural and solitary, and therefore appropriate for operation. The more localized the symptoms, the more hopeful is the prognosis. These tumours are usually fibromata with a vascular pedicle, lying loosely between the dura and the cord; or they may be fibrosarcomata with a good prognosis. The more widespread symptoms caused by a **gliomatous sarcoma of the spinal cord itself,** are very unfavourable. These symptoms vary with the position of the growth, and they partake of the character of a transverse lesion or of syringo-myelia. The only symptom which is really of value in this connection, is definite dissociation of the sensory disturbances, with the diminution of sensibility to pain and heat.

Operation has often revealed, instead of the expected tumour, *localized encapsuled collections of serous fluid* (meningitis serosa circumscripta) which might probably have been removed by simple puncture if the diagnosis had been possible (Krause, Oppenheim, Nonne, &c.).

(3) At Which Level is the Tumour Situated?

The accurate diagnosis of the level is an indispensable precedent of operation. Reference should be made to the remarks in connection with spinal cord injuries, and it is only necessary to add here that in practice the level which is diagnosed is usually too low. One should always fix upon the highest possible root which may be involved; but this may often be too low, and it will be necessary to search higher up at the operation.

CHAPTER LXXIX.

INFLAMMATORY DISEASES OF THE SPINAL COLUMN.

A.—TUBERCULAR CARIES.

TUBERCULAR caries is so much more frequent than any other form of inflammatory disease of the spine that, as far as the practitioner is concerned, it may be considered as the only important one. Difficulty in diagnosis only exists before the appearance of the characteristic symptom—sinking in of the diseased vertebra and the resulting deformity of the spine, the so-called Pott's curvature.

For purposes of diagnosis the disease is divided into several classes:—

(I) TUBERCULAR CARIES WITHOUT DEFINITE CURVATURE, AND WITHOUT A BURROWING ABSCESS.

This occurs more frequently in adults than in children, because the diseased vertebra soon softens in children, and the curvature develops early in a pronounced form. Nevertheless, a careful mother often seeks advice before the disease has reached the stage of deformity. The history and the method of examination varies with the age of the patient.

(*a*) If an *infant* is brought with the complaint that its entire behaviour has changed, that it is in marked distress, that it has become helpless, that it avoids any rapid movement of the body, that it cries even if lifted out of bed, although the mother is sure of not having hurt it, we should at once think of spinal disease. There is probably nothing to be seen on the back. At most, there may be a little rigidity, combined, perhaps, with some scarcely perceptible diffuse kyphosis, or at least with loss of the normal lumbar lordosis. A similar kyphosis is seen in rickets, but the spine remains movable in that disease, and the back at once makes a concave bend, if we swing the child, with its abdomen downwards, by its four extremities, as remarked by Hoffa. In spinal disease the vertebral column remains rigid, even in this posture, owing to the muscular fixation.

We might also be misled by Barlow's disease (scurvy-rickets) due to improper feeding. In this condition the child is also helpless, and cries when moved; but the pain is situated in the legs, and not in the back. Swelling and bluish-red discoloration of the gums in the vicinity of the erupted teeth indicate the nature of the disease.

(*b*) If the *child already walks*, we will be struck by the fact that it no longer plays with other children, and that it has difficulty in going up, and especially down, stairs.

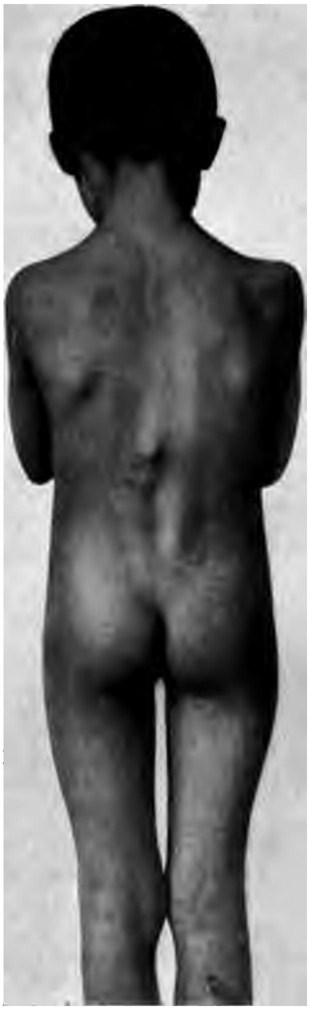


FIG. 281.—Early stage of spinal caries.

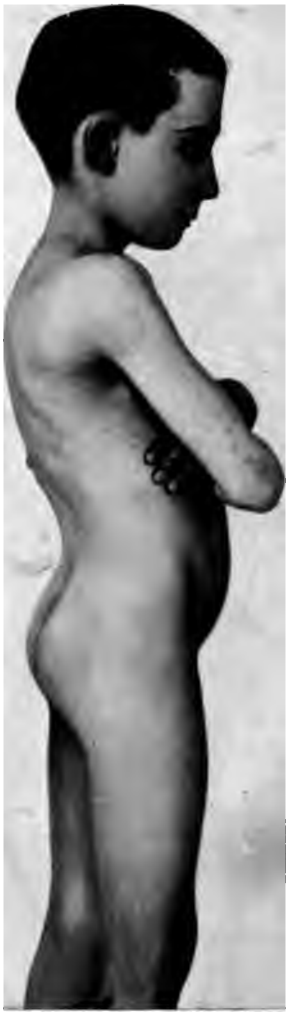


FIG. 282.—Same case, seen in profile.

On the other hand, children are sometimes seen with definite curvature, but without any subjective disturbances. This means that the acute process is over, that cicatrization and consolidation, which take place much earlier in children than in adults, have already occurred.

On examination, we are struck by the rigidity with which the spine is held, and by the way in which the child carefully avoids any movement of bending, over-straining, or rotating the spine. If the child is told to look round, he turns the whole body. If he has to get up from the floor, he behaves like a child with progressive muscular atrophy, i.e., he supports the hands on the knees. If we palpate the vertebral column, press on each spinous process separately, we shall be able to elicit pain at one definite spot. This also occurs if we press upon the spinal column in its long axis, obviously with care. If on repeated examination we are able to elicit this double form of sensitiveness to pressure, we are justified in assuming that caries has started — although it may be difficult to make the parents understand that some bone has already been eaten away, notwithstanding the striking mildness of the symptoms. It is necessary forthwith to explain the significance of the disease, otherwise it is impossible to secure the requisite careful treatment, and the patience which is demanded for it.

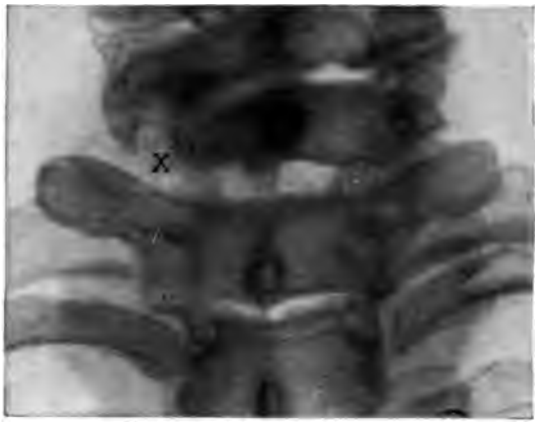
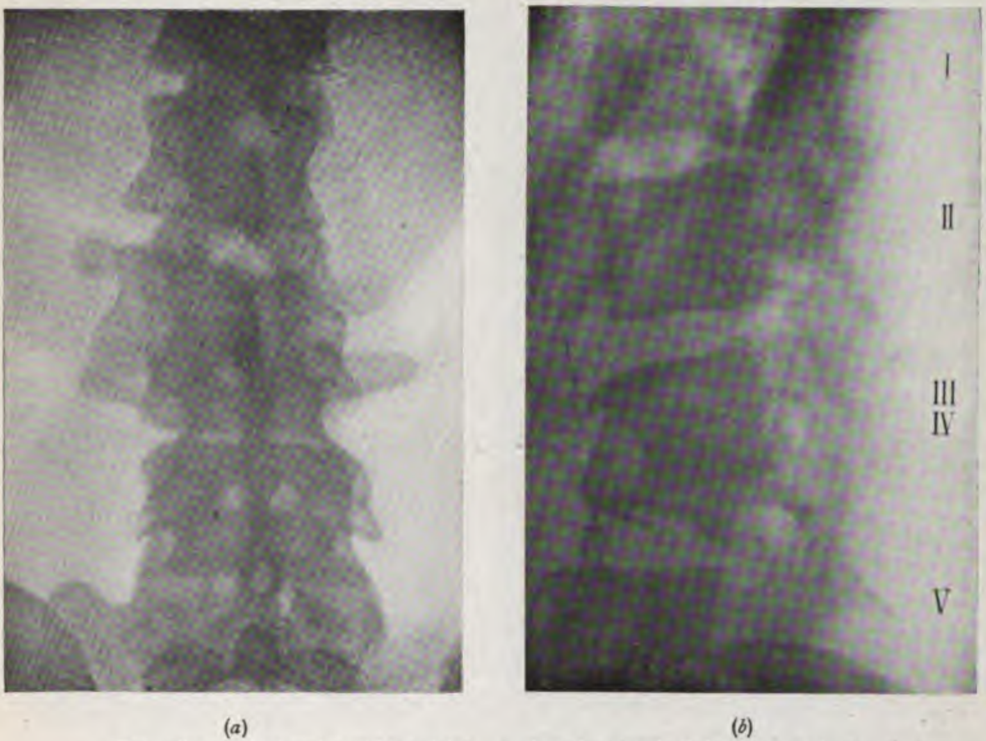


FIG. 283.—Early stage of caries in cervical spine. At X, edge of 7th vertebra eaten away.

(*c*) The previous history will be somewhat more ample in the case of *older children and adults.* We are, however, liable to be misled in these cases unless we have the possibility of spinal caries in view. Sometimes the patient localizes his pains to the umbilicus. More frequently we are consulted for sciatica, indefinite abdominal, or lumbar pains, intercostal neuralgia, "rheumatic" pains in the arms or back of the head. As long as these pains are unilateral, the diagnosis is often very difficult. But they almost always become bilateral after a few months, thus pointing definitely to disease of the spinal column. As previously stated, pain in the back on going downstairs, or knocking up against a stone on a level road, is quite pathognomonic.

An elderly, apparently vigorous, man became paraplegic without any apparent cause. On examination, the legs were found to be paralysed, and the bladder distended to the umbilicus. The shape of the spinal column showed nothing special, and the excellence of the general condition did not suggest any tubercular disease. The key to the condition was found in the patient's statement that he had felt for some weeks increasing pain in the back when walking about, when bending his spine, and especially when going downstairs. In such a case the question of a primary or a secondary new growth might also arise.



(*a*) (*b*)

FIG. 284.—Spinal caries with partial destruction of 3rd and 4th lumbar vertebræ. *a*, Anteroposterior view ; *b*, profile view.

The patient must be undressed for examination, and must stand with his knees close together, and be directed to bend his back forwards and backwards. If the movement is but slow and imperfect, and limited to the hip and knee joints, it must excite very grave suspicion, especially if the attempt to bend the spine backwards is a failure and causes the patient to groan. There can be no doubt about the diagnosis if, in addition, pain on axial pressure is present.

This latter test must be done very carefully, especially if the disease is in the cervical spine. We should not take the risk of making a patient paraplegic, or breaking off the odontoid process of the axis, in order to establish the diagnosis.

On pressing upon each separate spinous process, the detection of the diseased vertebra usually becomes very easy. Careful palpation and inspection of the patient in profile may probably reveal, even at this stage, some slight projection beyond the adjoining spinous processes (fig. 282), if this is not already evident from behind (fig. 281). In caries of the cervical spine there will also be some rigidity, and often some slight displacement of the head forward (widening of the neck in profile) before any definite curvature is evident (fig. 285).

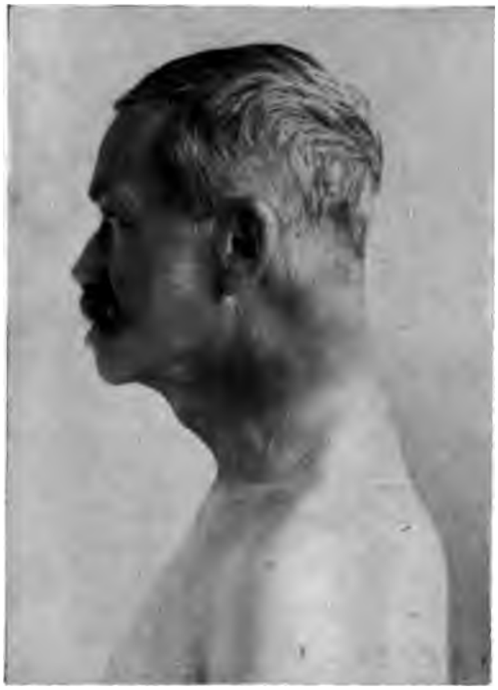


Fig. 285.—Caries of cervical spine (5th and 6th vertebræ). Head displaced somewhat forwards. Neck abnormally wide in profile.

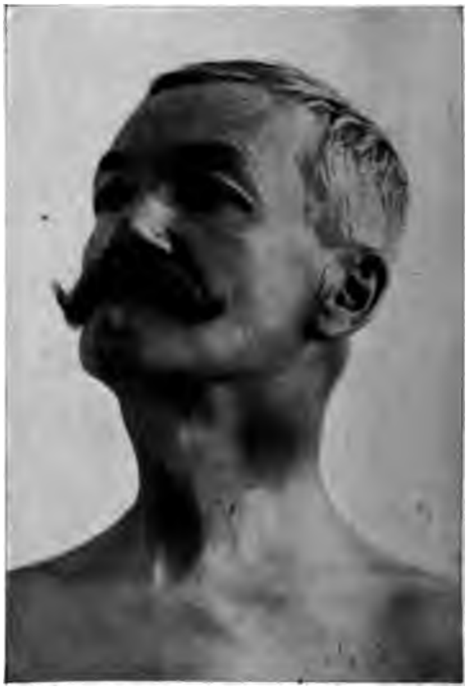


Fig. 286.—Abscess of neck due to caries.

The cases wherein these primary symptoms of pain on axial pressure and local tenderness are absent are more difficult to diagnose, especially if von Pirquet's reaction is feeble and some suspicion of hysteria exists. If the skiagram is inconclusive, the case may remain doubtful for months.

A vigorous young girl, aged 20, the picture of health, began to complain of lumbar pains, and wandered therewith from one hospital to another. The brother was tubercular. Caries was thought of, but the most careful examination failed to substantiate this view. The diagnosis remained in suspense until a burrowing abscess confirmed the suspicion. Even then there were no appreciable symptoms in the spinal column. The patient died from amyloid disease two years later.

(2) SPINAL CARIES WITH BURROWING ABSCESS.

In every case of spinal caries we must look for that common accompaniment of all tubercle of bone—**a cold abscess**—which is termed a **burrowing abscess,** because of its usual course. Its importance for diagnosis and treatment is evident from the fact that it is present in at least one-fourth of the cases—according to other statistics, one half.

There are cases wherein a burrowing abscess is the first, and for a long time the only appreciable sign of spinal caries. Before we discuss its diagnosis, we will briefly refer to its anatomical relations.

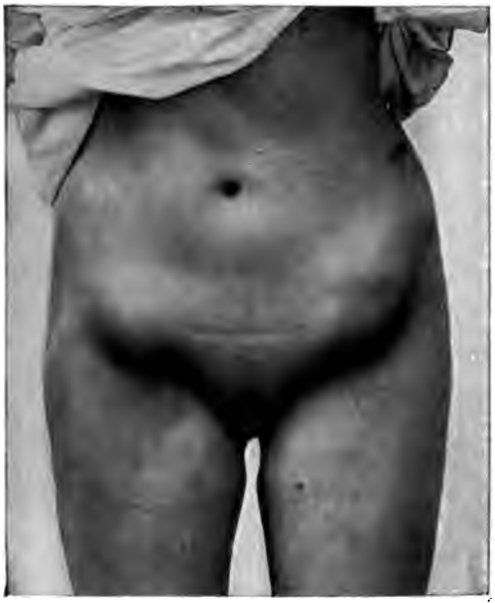


FIG. 287.—Caries of 4th lumbar vertebra, with bilateral inguinal burrowing abscess.

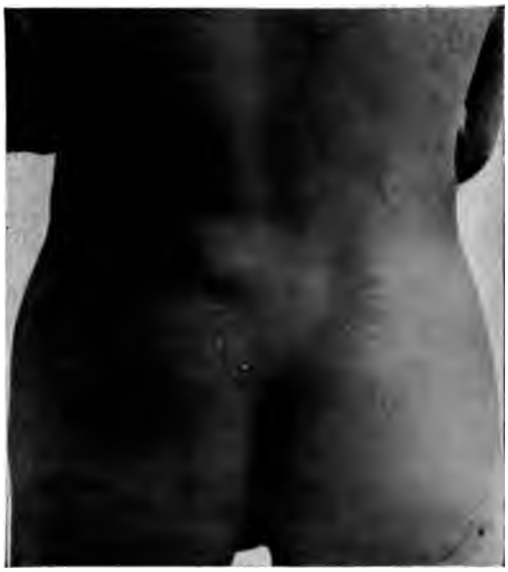


FIG. 288.—Commencing abscess in tubercle of left sacroiliac joint.

In caries of the *upper cervical spine* it is found in the posterior pharyngeal wall, or, more frequently, at the side of the neck, in front of or behind the sternomastoid. Exceptionally it may run under the clavicle towards the axilla. If the caries affects the *lower cervical spine,* and the abscess originates in the transverse process or the vertebral arch, it may also run under the muscles of the back, but as a rule it tracks along the œsophagus, penetrates the thorax, and behaves like abscesses which are derived from the *dorsal vertebræ.* The latter reach the surface between the twelfth rib and the ilium, or they dive down deeply, following the large vessels over the ileo-psoas muscle as far as Poupart's ligament, and eventually burst somewhere through the muscles and reach the surface.

If, in lumbar caries, the diseased focus is in the body of the vertebra, the abscess burrows in front of the spine, in the sheath of the psoas, travelling downwards either over or under Poupart's ligament, to appear in the inguinal region or in the anterior femoral triangle. The abscess appeared in the former position in the case illustrated in fig. 287. Despite the two burrowing abscesses, there was neither curvature nor definite disturbance of function at first; nothing but slight tenderness on pressure over one spinous process. The skiagram showed clearly that the fourth lumbar vertebra was affected.

More rarely the abscesses follow the hypogastric artery and the sciatic nerve, traverse the great sciatic foramen, run under the gluteal muscles, and sometimes from there proceed to the posterior surface of the thigh.

If the focus of disease is situated in the *laminæ* or in *the vertebral arch*, the abscess appears on the back.

The same applies to foci of disease which are situated posteriorly in cases of *tuberculosis of the sacrum*, or of the *sacroiliac joint*. But if the disease is situated on the anterior surface of this joint, the pus gains access to the sheath of the iliacus muscle, fills the side of the pelvis, and may burrow thence under Poupart's ligament into the thigh (fig. 289, so-called iliac abscess). It is then situated either at the side of, or beneath the sartorius. If the focus of the disease is more deeply situated, the abscess burrows towards the perinæum and makes its appearance there as a perirectal abscess.

If we have already ascertained the correct diagnosis by means of signs in the vertebral column, there is no difficulty in understanding the significance of a burrowing abscess. But if this is the first symptom which the patient notices, and for which he consults the doctor, many errors of diagnosis are possible, as already mentioned in detail, unless a careful examination is made.

We may summarize these once again for the purpose of taking a rapid view of the position.

In the *neck* one might think of a deep lipoma, a deep branchial-cleft cyst, or of an œsophageal diverticulum. Burrowing abscesses have even been mistaken for goitres.

In the *thorax* one should think especially of lipoma, cold abscess originating in the rib, or a pleural effusion which has spontaneously broken through.

In the *lumbar region* one should think of lipoma, lumbar hernia, caries of the ribs or pelvis, and also of the possibility of a tubercular perinephritis, which has burst through posteriorly. The urine must, therefore, be examined in every case of lumbar abscess.

An abscess of the pelvic fossa may, if on the right, be mistaken for an ileocæcal tumour; if on both sides, for caries of the pelvis or chronic pelvic osteomyelitis, or even for pelvic tumours. The correct diagnosis depends upon the accurate observation of the superior connections of the swelling. Very frequently flexion of the hip-joint is produced, and thus spinal caries may be mistaken for hip disease. The pelvic cavity must, therefore, be examined as thoroughly in what is apparently hip disease as in spinal caries.

Abscesses of the pelvic fossa, as we have already seen, consist of iliac and psoas abscesses. When there is extensive suppuration, it is not possible to draw a sharp distinction between the two forms, nor is it important to do so. The occurrence of clinically primary suppuration in the psoas muscle is exceedingly rare, and then it is generally to be attributed to some trauma. This condition might justify the popular old term "psoas abscess" as a separate disease.

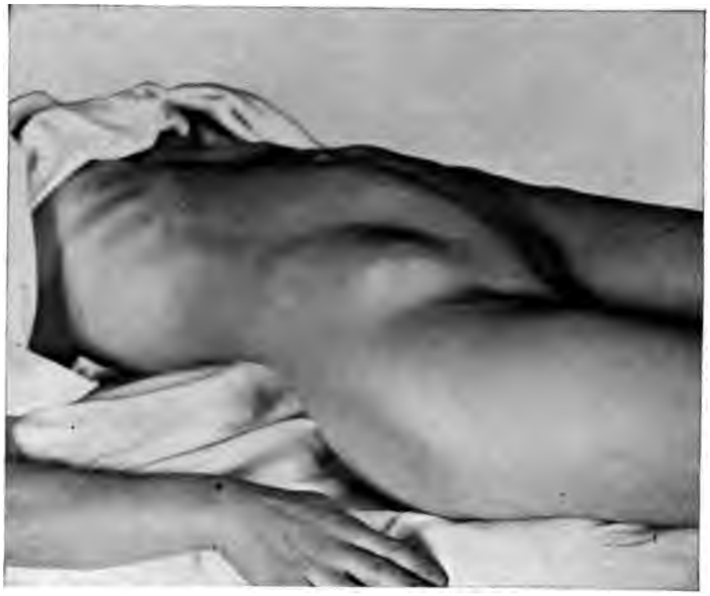


FIG. 289.—Burrowing abscess in the iliac fossa in a case of sacroiliac tuberculosis.

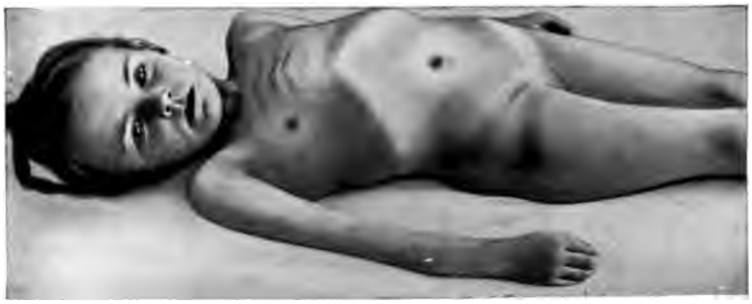


FIG. 290.—Caries of the dorsal spine with a transversely divided burrowing abscess sac.

Inguinal abscesses have been mistaken for inguinal hernia and hydrocele of the canal, especially in women. But these abscesses are situated more towards the side, and have a wide connection with the pelvic bone by broad processes, so that this mistake should be avoided; the very rare bilocular hydroceles may, however, still give rise to difficulty.

Abscesses of the thigh may be mistaken for femoral herniæ, if situated in the middle line immediately under Poupart's ligament; if towards the side, for hygroma of the subiliac bursa. If the pus can be displaced at all, it goes back *gradually*, a hernia goes back with a jerk. If it is not displaceable—this is the rule—its consistence is generally elastic or fluctuating, which, of course excludes a hernia. An enlarged bursa is recognized by its deep situation under the iliacus, whereas an abscess, even if it descends within the sheath of the muscle, always has a tendency to reach the surface. Many burrowing abscesses are distinguished by possessing a subdivided sac (fig. 290). If the femoral abscess is situated lower down, it may be mistaken for sarcoma of the femur, or of the adductor muscles. The flexion of the hip-joint may sometimes suggest hip disease.

Unless the hip-joint is itself secondarily affected with tubercle, a burrowing abscess due to spinal caries only prevents extension, whereas in true hip-disease abduction and rotation are especially limited.

In *perineal abscesses* the diagnosis has to be made from dermoids and the various forms of periproctitis. The most likely diagnosis in the rare cases of *gluteal* burrowing abscesses is hip disease, which can only be excluded by a careful investigation of the spinal column and the hip-joint.

The origin of doubtful cases of suppurating fistulæ is best ascertained by means of a skiagram, after they have been injected. The most useful preparation for this purpose is one composed of vaseline with 20 per cent. of zircon oxide, a modification of Beck's methods, as bismuth salts are not quite free from risk.

(3) SPINAL CARIES WITH CURVATURE.

Once the typical curvature is developed, a glance at the patient's back suffices for the diagnosis. It is usually very easy to avoid any confusion with spinal deformities due to other causes. As the kyphosis in spinal caries depends upon the destruction of one, or at most of two or three, vertebræ, it appears more or less in the form of an *angular kink*, the apex of which is easily recognized to be formed by *one* spinous process. All other changes in the shape of the spinal column, except those which result from accidents, are not merely kinks but *curves*, because they concern several vertebræ. There should never be any confusion with scoliosis and kyphoscoliosis, because lateral curvature is so predominant in these, but is only very exceptionally present in tubercular disease. Even if a vertebra is diseased asymmetrically, as occurs occasionally, and therefore breaks down more on one side than on the other, there is no real scoliosis, but always an essentially anterior kink, a gibbus. In rachitic bending of the spine, the result is more frequently pure or nearly pure kyphosis, and the deformity consists of a definite *curve* and not of ***kink***. If a child, suspected of caries, is *carefully* suspended, by all its limbs, with the abdomen downwards, the back will bend in rickets, but not in caries.

(4) SPINAL CARIES WITH CORD SYMPTOMS.

If a patient with spinal caries becomes affected with *spastic paraplegia,* it obviously indicates compression of the cord, but simple loss of power of gait is sometimes ascribed to the vertebral disease, although it is really the result of an early spastic paraparesis. We may even go further; any definite *increase in the tendon reflexes,* in the parts supplied from below the lesion, must be regarded as a sign of commencing pressure. Sometimes *symptoms of pain* predominate; occipital neuralgia in "malum sub-occipitale," neuralgic pains in the arms in caries of the lower cervical vertebræ, crural neuralgia and sciatica when the lumbar spine is affected. These symptoms point to compression of the roots and should prepare us for the rapid onset of cord symptoms, or compel us to try to avoid them by means of permanent extension. The absence of dislocation of a vertebra in no way negatives the diagnosis of damage to the cord, which is often involved in an actual extension of the disease to the membranes, and is not compressed by displacement of the vertebræ. In such cases there is, of course, but little to be expected from extension treatment.

If intercostal pains are present in a case of curvature, it is usually assumed that the curvature cannot possibly be due merely to rickets or to habit—but this assumption is unjustifiable. There are cases of scoliosis or kypho-scoliosis, wherein the intercostal nerves are nipped between two ribs with the production of severe neuralgia. Rest in bed and extension rapidly bring relief.

B.—NON-TUBERCULAR INFLAMMATION OF THE SPINAL COLUMN.

We have, hitherto, been assuming that the spinal disease is of a tubercular nature, which, as a matter of fact, is true for the majority of cases. But there are various exceptions to the rule.

Certain changes in the spine, for instance, follow **injuries**, and the significance of these has loomed large since the prevalence of insurance against accidents.

An elderly man rolled down a small incline in his garden. He felt some pain in the lower portion of his cervical spine, but only kept his bed for a short time. Later on, his cervical spine began to bend forwards, and there developed a curvature which was something between an angular kink and a roundish kyphosis. There was no burrowing abscess, no pyrexia, &c., and the vertebra which formed the apex of the curve was not tender on pressure.

Did the patient suffer from tubercular caries of a traumatic nature, brought on by his fall? It is possible, but experience shows that a similar clinical picture may develop without any tubercular change. The comparatively short period, during which pain was complained of, is very unlike tubercle. It is much more likely that the patient sustained a *compression-fracture* of the spine, and that the damaged vertebral body gradually broke up and sunk in. This condition is termed "traumatic spondylitis."

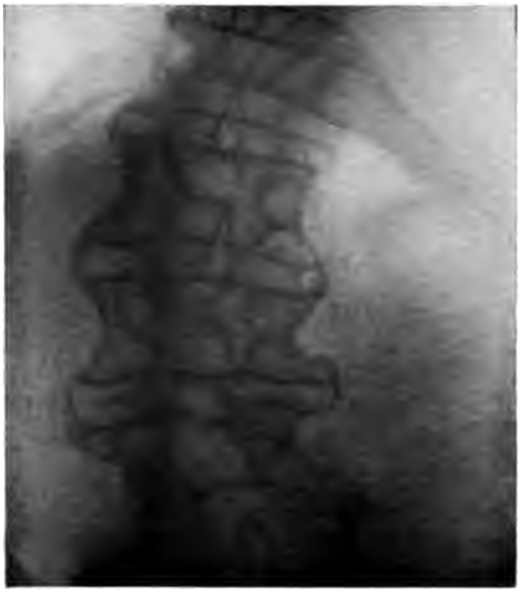


FIG. 291.—Arthritis deformans of the spine.

The difficulty of diagnosis is very great in a case where there has been a slight initial injury, and the progress of the case eventually reveals a burrowing abscess and destruction of bone, proving that tubercle is responsible. The question arises whether the tubercle was originally excited by the injury or whether it was merely made manifest by it. A careful history will often demonstrate that symptoms were present before the injury occurred.

The vertebral joints are subject to the varied pathological processes which attack other joints, and produce proliferation, deformity or ankylosis. But, in contrast to tuberculosis, these processes usually affect a large portion of the vertebral column. If the upper dorsal vertebræ are affected, the kyphotic posture is striking, and if the lumbar vertebræ are affected the vertical rigidity becomes marked. Sometimes the vertebræ suffer quite alone, at other times other joints —especially the larger ones—are simultaneously affected, with a subacute or chronic rheumatism, subject to exacerbations, which eventually lead to ankylosis.

Disturbances of the nerve roots have been observed in a few cases. (Bechteren, Strümpell, Pierre Marie). Our clinical and pathological knowledge is still too deficient to attempt any classification of these cases.

Gummatous periostitis in **tertiary syphilis,** by destroying the affected vertebræ, may produce a clinical picture very similar to spinal caries. The diagnosis must be based on the history, or on Wassermann's test, and on the result of specific treatment.

Finally, one should mention the rare occurrence of **acute osteomyelitis** of the spine, and the metastatic inflammation of the spine that has been observed after acute infective diseases, such as **pneumonia** and especially **typhoid fever.** The condition has even been known to follow a boil. The course of these diseases depends upon the virulence of the organisms. It is the duty of the practitioner to detect abscess development as early as possible, but this does not, however, occur in all cases.

CHAPTER LXXX.

SPINAL CURVATURES.

ORTHOPÆDICS has, of late, become so much of a speciality, that not only the general practitioner but even the surgeon gladly leaves spinal curvature to this department. Expedient as this may be for therapeutics, because only an orthopædic institute can possess all the mechanical apparatus for the treatment of scoliosis, it is not so as far as diagnosis is concerned. To detect the beginnings of scoliosis, nothing is required beyond an observant eye and a plumb line. Nevertheless the strictures applied by Albert years ago, in regard to overlooking scoliosis, are equally applicable to-day. Perhaps lady doctors would have the advantage in the diagnosis of this condition, because females possess a keener eye for deformity than males. It is for this reason that the mother first detects that the "child has one shoulder higher than another," or that "the back or hip sticks out."

In considering the *causes* of spinal curvature, we must recollect, as Schulthesz has especially pointed out, that it is not a *clinical and etiological entity*, but is in most cases only a *symptom.* When the statics of the body are disturbed at any point, the spinal column provides for the restoration of the equilibrium. Shortening of one leg causes obliquity of the pelvis, and a corresponding scoliosis occurs to compensate for this disturbance (figs. 292 and 293). An abnormal inclination of the pelvis, due to flexion of the hip, is compensated for by an increased lumbar lordosis. These are *static curvatures*, and are recognized by their disappearance as soon as the pelvis is restored to its natural posture (figs. 292 and 293). Temporary curvatures are often due to some painful condition, the best example of which is sciatica (fig. 294), a matter to which we shall again refer. In other cases the curvatures are due to disturbances in the supporting power of the *muscles*. Thus we find considerable lumbar lordosis in progressive muscular atrophy (fig. 295); scoliosis in anterior polio-

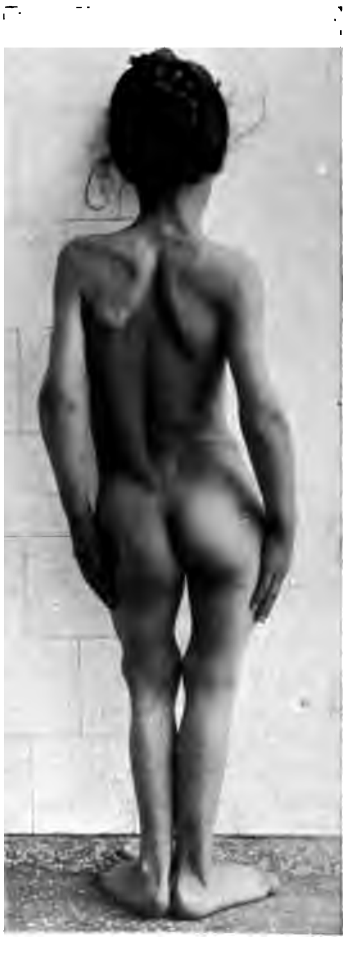


Fig. 292.—Lumbar scoliosis with convexity to the left, due to 4 cm. shortening of left leg.

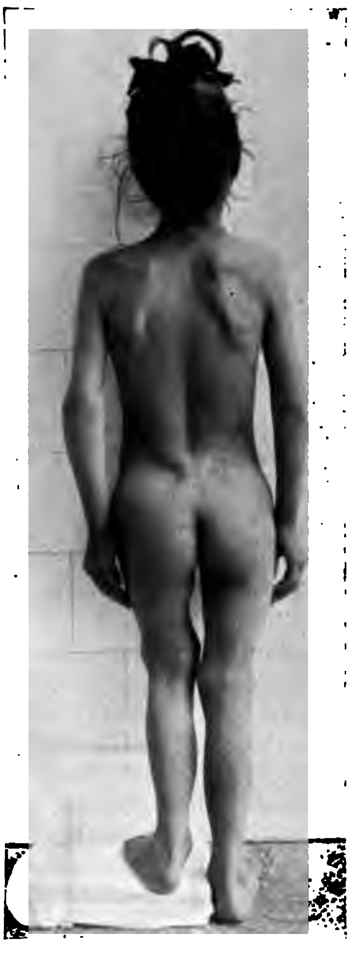


Fig. 293.—Same case, after compensating for the shortening.

myelitis, in syringomyelia, and in Friedreich's disease (fig. 296). *Diseases of the thoracic organs* sometimes are at fault, as in the case of scoliosis which occurs in consequence of contraction after pleurisy, and especially after empyema. Heart disease with enlargement, by causing asymmetry of the thorax, may also lead to curvature of the spine.

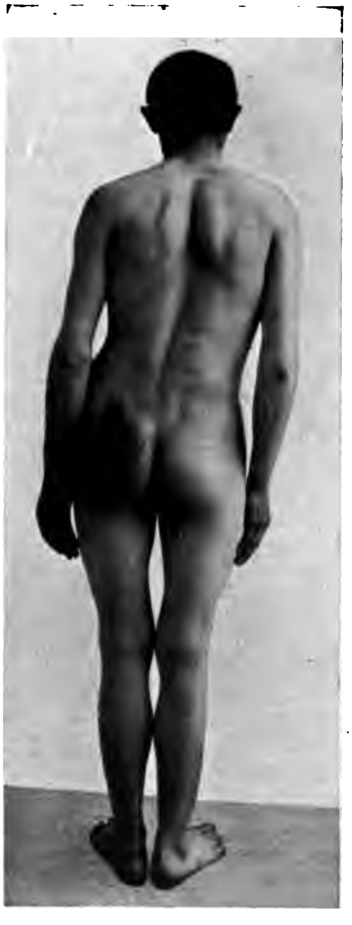


FIG. 294.—Scoliosis due to sciatica.

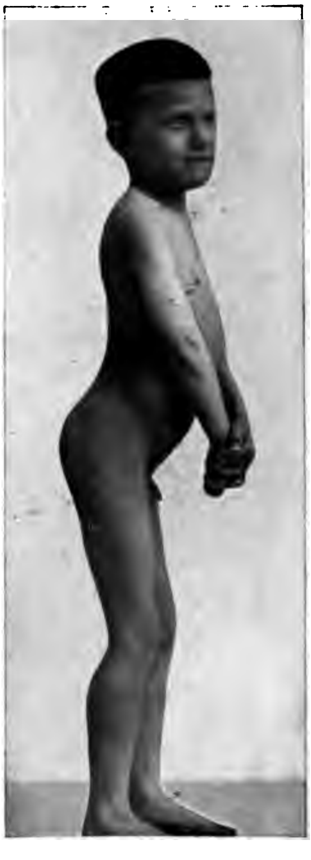


FIG. 295.—Progressive muscular atrophy with lordosis.

We should only look in the *spinal column itself* for the origin of the trouble, if we have excluded all these causes. *Inflammatory diseases* play their part among these spinal changes. Caries leads mainly to kyphosis, but occasionally produces a slight lateral curvature. Arthritis deformans of the lumbar spine may also cause lumbar kyphosis. Injuries may be responsible, for sometimes kyphosis is caused by a compression-fracture.

Finally there is a group of cases in which the change in form is due to a *congenital asymmetrical deformity of the spine*, such as a wedge-shaped outgrowth of a vertebra, with a supernumerary rib on the broad side, or fusion of two vertebræ on one side, with the absence of a rib on that side. If the cervicodorsal portion is affected, the deformity manifests itself by one shoulder "*standing out*" (fig. 297). This congenital prominence of a shoulder may also be due to simple muscular anomaly, or to a clasp-like bony communication between the scapula and the cervical spine A skiagram is always required to elucidate these conditions. These deformities are mainly responsible for the so-called "numerical variations" of the spinal column, i.e., they interfere with the ordinary subdivision of vertebræ and ribs into the separate segments (Dwight, Böhm). It still remains to indicate

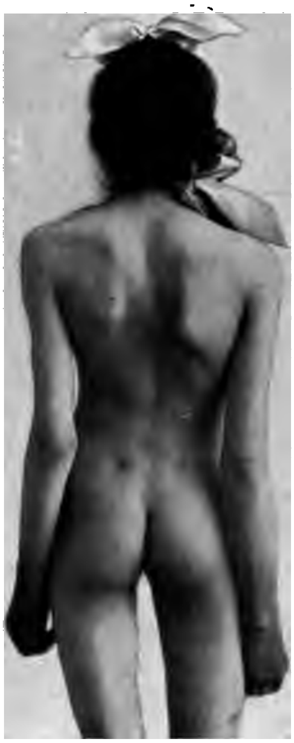


FIG. 296.—Friedreich's ataxia with scoliosis.

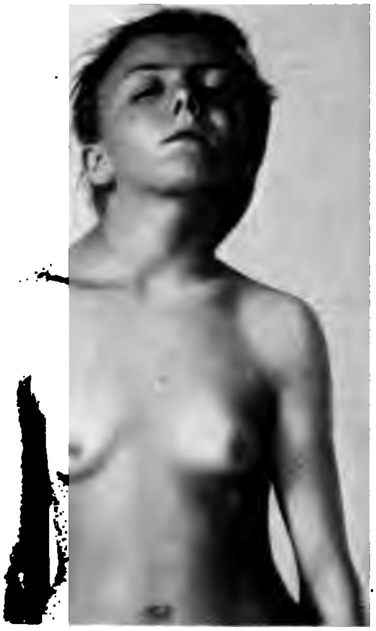


FIG. 297.—Congenital cervicodorsal scoliosis with convexity to the right, and prominence of the shoulder, due to an asymmetrical outgrowth of spine and ribs.

those forms of slight asymmetry which are responsible for ordinary scoliosis.

The further consideration of this subject comprises that which is usually understood by the term "*curvatures of the spine.*"

These curvatures are (1) symmetrical or antero-posterior, (2) asymmetrical or lateral.

(1) ANTERO-POSTERIOR CURVATURES.

These may either consist of an **abnormal flatness,** the bootmaker's type of spine, or of an increase in the normal curves, or, finally, of really abnormal curvatures. The increase of the normal curve in the lumbar region constitutes **lordosis** and in the back **kyphosis.** The mechanical and the nerve conditions already referred to are the principal causes, but rickets and osteomalacia should also be especially considered. Then there is the so-called "round back" which is hereditary. Such abnormal curvatures as **lumbar kyphosis** usually depend upon rickets or osteomalacia.

The diagnosis of these antero-posterior curvatures is easy. It only requires some slight appreciation of form, and we will therefore not dwell on it any longer.

It is only necessary to remark that one must examine for lateral curvature in all antero-posterior curvatures. Slight scoliosis with definite torsion is often concealed behind an ordinary round back.

(2) LATERAL CURVATURES.

Lateral curvatures, or **scoliosis,** involve the practitioner in great responsibility, because they generally bring their own revenge for delayed treatment.

The patient should stand for examination, completely nude, or at least undressed as far as below the hips; the two feet must be held in the same posture, the arms must hang down loosely, and otherwise the whole attitude must be as unconstrained as possible. Then we look at him from the back, making an inspection from head to foot. We note whether the head is exactly over the mid-point of the feet, whether it is held obliquely or erect, whether the shoulders are at the same level, whether the scapulæ are at equal distances from the spine and stand out equally from the thorax. Our eyes follow the line of the spinous processes, and we can at once detect, especially in thin subjects, whether the furrow between it and the transverse processes is equally developed on both sides, or whether it is somewhat obliterated on one side and deepened on the other. We compare the distance of the arms from the body, in other words the two triangles of the waist, i.e., the triangle formed by the arm with the outline of the body, and also the shape of the thorax, the position and shape of the hips, the level of the gluteal folds, and finally the shape and posture of the legs. Then the patient is directed to walk a few steps, in order to see whether he limps. After he has walked round the room, we tell him to resume his former position. This slight interruption in the examination has the advantage of showing us whether the posture observed at first is really the normal posture of the patient. This is a precaution which should not be omitted even in slight cases of scoliosis which can easily be straightened out, and especially not in cases of complete scoliosis (figs. 311 and 312). We next proceed to a more careful examination of the spinal column. We drop a plumb line (fig. 298) from the seventh cervical spine, and note whether it falls midway between the buttocks, and lower down between the feet. We thus find to which side the thorax is displaced, and the extent of the displacement in relation to the pelvis, and also the distance by which the convexity of the curvature deviates from the plumb line (figs. 298 and 299). We then feel the spine and mark on the skin the position of each spinous process with a pencil. This also gives the opportunity of observing whether there is any tenderness on pressure. We then proceed to test the movement of the spine, and we direct the patient to bend his back forwards, backwards, and to each side, with his knees close together. This examination, in the first place, will reveal any caries which may have been overlooked, for the movements are but slightly restricted and never painful in scoliosis, whereas caries may always be recognized by painfulness and the limitation of movement by pain which bears no relation to deformity. But the examination of the back, when bending forward, teaches us something more. On inspecting the bent back from the nape of the neck downwards, we may be struck with the fact that one side of the thorax is higher than the other, that is to say, that there is protuberance of the ribs (figs. 302 and 303). This permits us to estimate the degree of torsion of the vertebræ, and the amount of the deformity. We can, however, usually detect the protuberance of the ribs when the patient is erect.

The inexperienced will, however, be astonished to see how pronounced it really may be, when the back, in the erect posture, appears to be fairly normal to the unpractised eye. Neither should we estimate the degree of change exclusively by the position of the spinous processes, because they always remain nearer to the middle line than the bodies of the vertebræ. The deformity is always more noticeable on the skeleton from a front view than from the back, and the vertebral spine may have undergone a pronounced lateral deviation before any marked displacement of the spinous processes can be detected.

The examination of the functions of the spinal column indicates, finally, the *stage* of the deformity. If all the movements are carried out symmetrically to the same extent, and if the deformity vanishes with the movements, or if it is only noticed occasionally, for instance, when the muscles of the back are tired, the case is in an *early stage*, wherein no material changes in the spinal column have yet developed. Under appropriate treatment, this condition may be cured in a few weeks. If, however, the deformity is present in the same manner at every examination, but can be completely straightened out, actively by

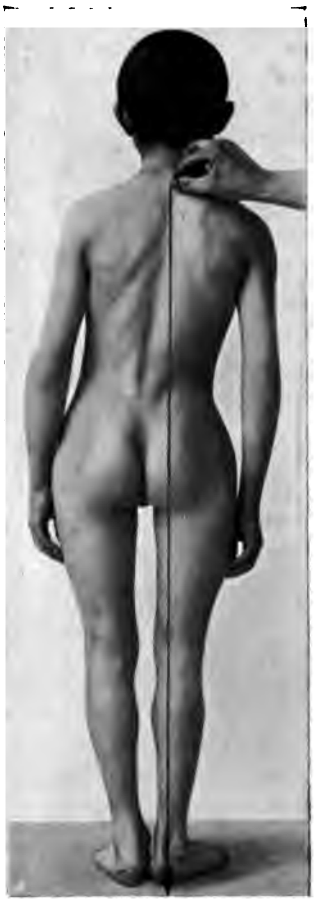


FIG. 298.—Rachitic, lumbar scoliosis with convexity to the left, and displacement of thorax to right, i.e., to the side of the concavity (through over-compensation).

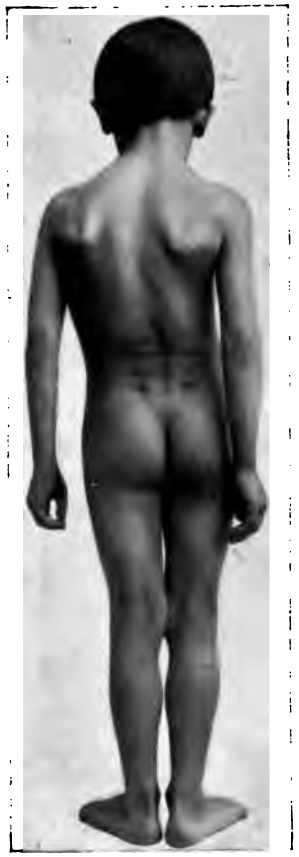


FIG. 299.—Dorsal scoliosis with convexity to left, and displacement of thorax to left, i.e., to the side of the convexity.

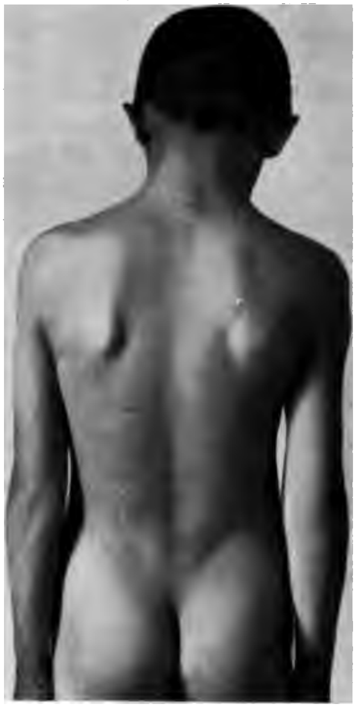


FIG. 300.—Complete scoliosis with convexity to left, displacement of thorax to right. Right waist triangle enlarged. Right axillary fold shortened.

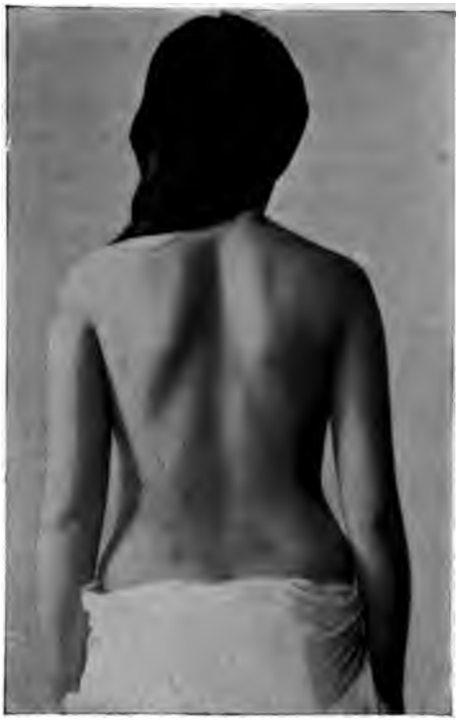


FIG. 301.—Lumbar scoliosis with convexity to left, of very slight degree; only recognizable by shape of waist triangle.

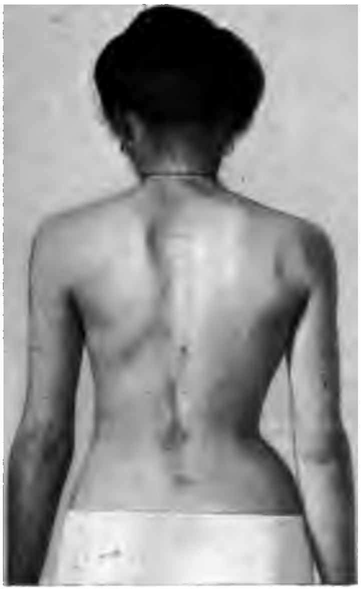


FIG. 302.—Lumbar scoliosis of severe degree with convexity to the left and dorsal scoliosis with convexity to the right. Despite an apparently slight curvature of the spine, the right waist triangle is deeply indented.

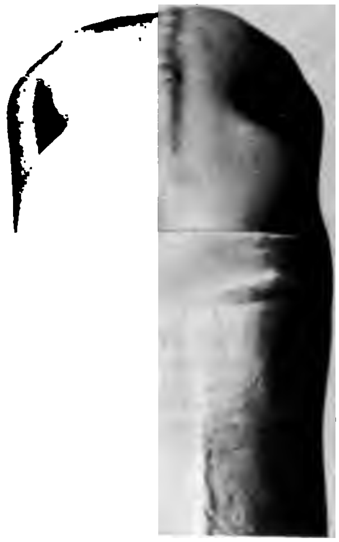


FIG. 303.—Same case as fig. 302. Pronounced protuberance of ribs on right side, despite an apparently slight lateral curvature.

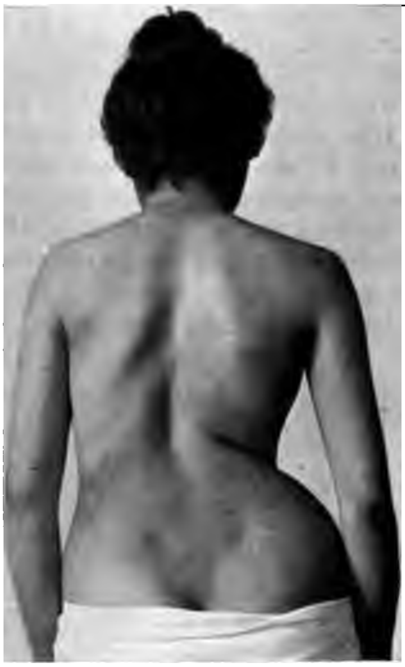


FIG. 304.—Severe lumbo-dorsal scoliosis, with convexity to the left and compensatory dorsal scoliosis with convexity to the right.

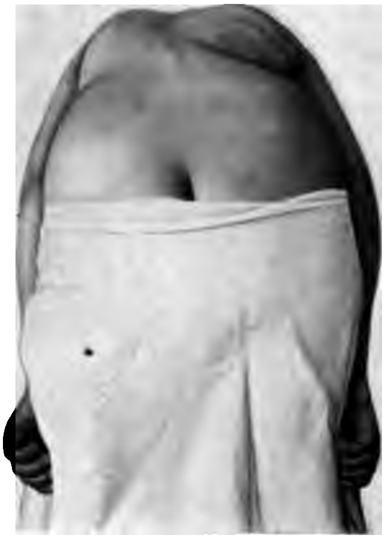


FIG. 305.—Same case as fig. 304. Lower protuberance of ribs to the left, upper protuberance to the right.

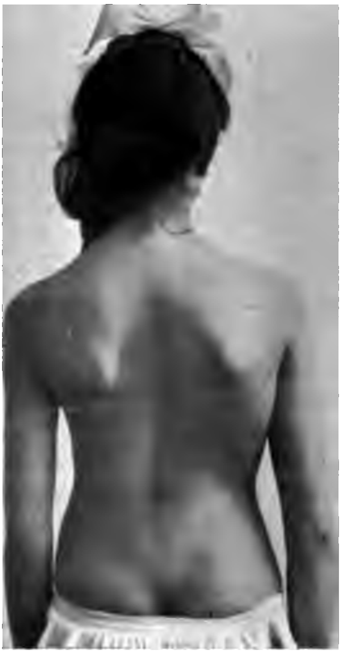


FIG. 306.—Primary dorsal scoliosis with convexity to the right. Mild case.

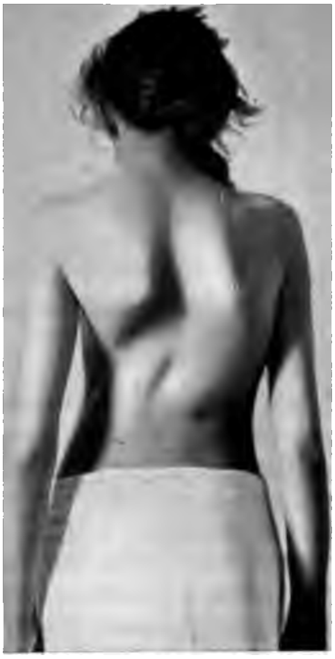


FIG. 307.—Primary dorsal scoliosis, with convexity to right. Severe case.

muscular exertion or passively by suspension, the case is one of *mobile scoliosis* (figs. 311 and 312). Immediate treatment promises good results even here. We must not be content, however, with prescribing a tonic and giving general directions that the patient must not tire himself. We must either take the treatment in hand ourselves, energetically—and complicated apparatus may not be required in this stage—or we must send him to an orthopædic institute. If the deformity can only be partially straightened out, it is in a *position of contracture*; if it cannot be straightened out at all, the case is one of *fixed scoliosis*.

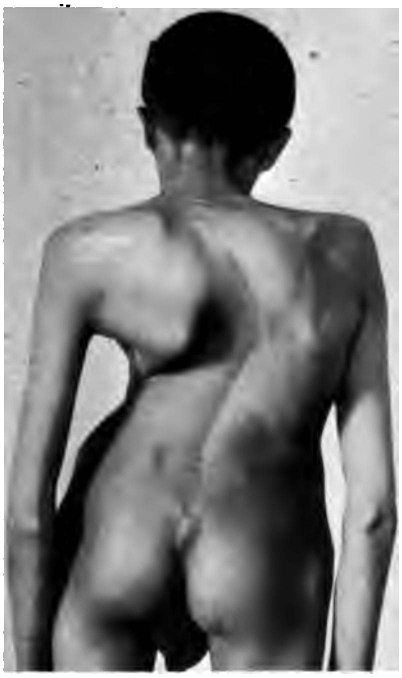


FIG. 308.—Primary dorsal scoliosis with convexity to the right. Very severe case.

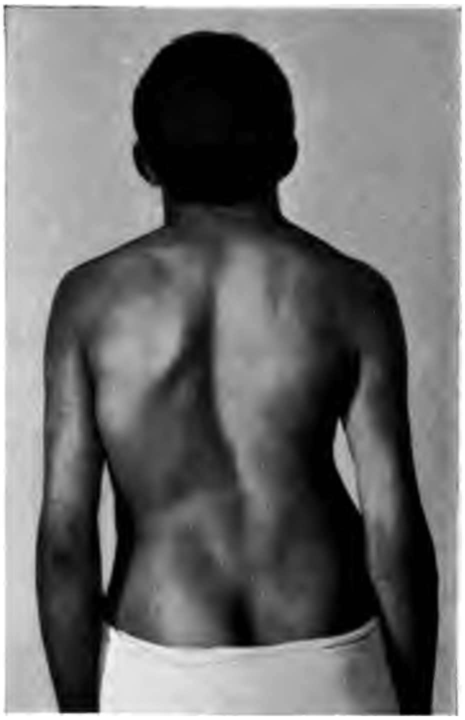


FIG. 309.—Primary dorsal scoliosis with convexity to the left.

We now have to consider *the form* of the scoliosis. If the whole spine constitutes one curve, we speak of **complete scoliosis,** which is usually *convex to the left.* It is recognized by the fact that the waist triangle on the convex side is diminished with sharp angles above and below, whereas on the concave side the triangle is enlarged and more deeply indented (fig. 300). In early cases the back is usually displaced to the left in relation to the pelvis.

In complete scoliosis the protuberance of the ribs is generally found on the concave side, i.e., towards the right, in contrast to the condition in partial scoliosis.

If the curvature is mainly localized in the lumbar region, we speak of *lumbar* or *lumbo-dorsal scoliosis*. Here also the convexity is usually to the left (figs. 298, 301-305). The displacement of the back in relation to the pelvis is more pronounced than in the previous form, the waist triangle on the convex side is also diminished or filled out, the protuberance of the ribs is on the convex, i.e., the left side. In other cases the shape of the waist triangle is the only indication of the curvature (fig. 301). This form has a great tendency to be followed, in the course of time, by a compensatory curvature of the dorsal vertebræ in the opposite direction, i.e., towards the right, and, of course, a second protuberance of the ribs develops on the right side. Fig. 305 illustrates this double protuberance of ribs in a severe case of dorsal scoliosis with the convexity to the right.

This must be distinguished from **primary scoliosis of the dorsal vertebræ,** wherein the curvature has its *convexity to the right* (figs. 306-308, 310), and which eventually becomes compensated by a lumbar scoliosis with its convexity to the left. The whole of the upper part of the trunk appears to be displaced to the right in relation to the pelvis. The right arm hangs free of the body, whereas the left is closely applied to the hip. The right scapula appears to project, because it is pushed forward by the protuberance of the ribs. On the other hand, the left scapula is really farther away from the body than the right one, because it lies over the flattened or retracted half of the thorax.

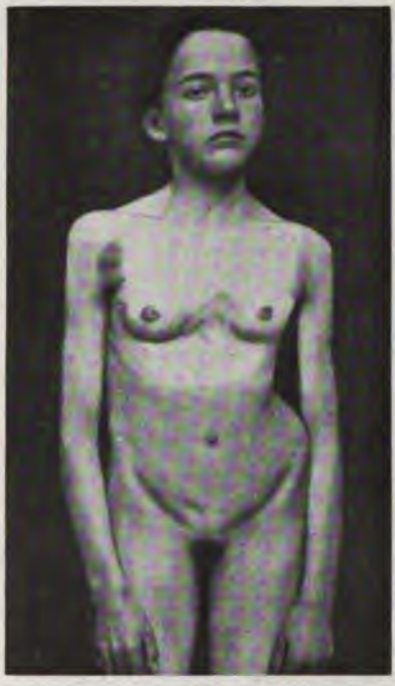


FIG. 310.—Dorsal scoliosis with convexity to the right. Displacement of upper part of trunk to the right.

Having correctly ascertained the form of the curvature, our next task is to discover its *cause*. There is no difficulty in this if we are told by the mother that the child was late in walking and in talking, and that the teeth were late in appearing, and if we also find evidences of *rickets* still persisting in the skeleton. We may also obtain some indications from the form of the rib protuberance, for if the ribs are bent close to the spine this points strongly to rickets. Sometimes there are no indications of definite rickets, but other abnormalities of the skeleton, such as flat-foot or knock-knee, show that the cause must be ascribed to *late rickets.* In other cases, the examination of the brothers and sisters, or even a glance at the mother's back, may show that there is a *hereditary tendency*, which cannot be identified with rickets.

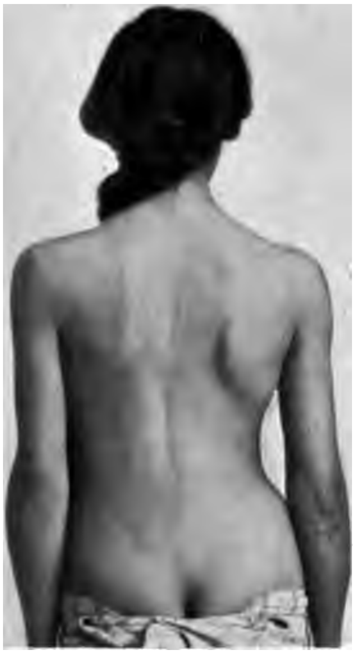


FIG. 311.—Mobile complete scoliosis with convexity to the left. Muscles relaxed.

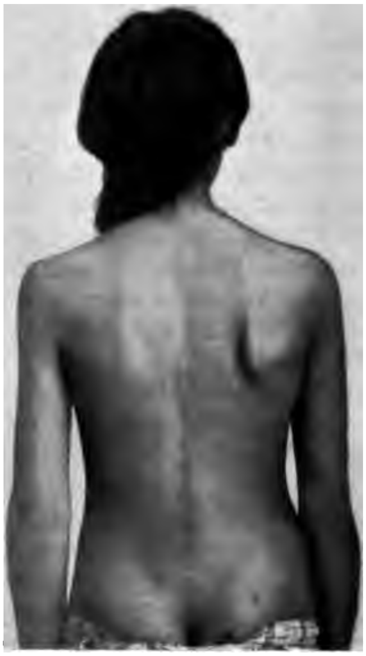


FIG. 312 Same case, with the muscles tense.

Finally, what is our position in regard to *school scoliosis?* There can be no doubt that the most frequent types of lateral curvature are commonest among school children, viz., lumbar or complete scoliosis with the convexity to the left and dorsal scoliosis with convexity to the right. On the other hand, we often see these types develop at an age before it is possible that the posture adopted in writing could have any effect. We must, therefore, assume that the scoliosis produced in school develops on some pre-existing tendency thereto. Not only do the cramped posture of writing, the position of the copy-book, and the shape of the letters contribute towards the development of the curvature, but also the bad habit of always carrying the school books home under the same arm. Worse than all this, however, is the practice, which was universally prevalent up to a few years ago, of making children sit up straight for hours on forms without adequate supports for the back. No wonder that weak muscles of the back become tired and the spine sinks into a posture to which a pathological predisposition inclines it.

This predisposition probably depends upon an exaggeration of some slight asymmetry of the spine, normally present. But this must not be classed, without further consideration, with the condition previously referred to as "numerical variation," depending upon an essential mal-development, the significance of which is not quite clear.

PART VI.

SURGICAL DISEASES OF THE UPPER EXTREMITY.

CHAPTER LXXXI.

FRACTURES AND DISLOCATIONS OF THE CLAVICLE.

If a patient comes to consult us, after an accident, with his arm in a sling, because he cannot use it, our first thought will be of the shoulder girdle, and more particularly of the clavicle which is most susceptible of injury. Injuries in this region may be classified into three groups.

(1) If a patient is unable to raise his arm beyond the horizontal, after a fall on the shoulder or a fall on the arm which transmits its force to the shoulder, if he inclines his head towards the injured side (fig. 313), and if the shoulder appears to be drawn forwards and inwards, we involuntarily look to the clavicle, in the anticipation of finding a fracture in its outer half (fig. 313). The patient suffers extreme pain when the fracture is manipulated. But this striking picture is not always in evidence. In children especially, the fracture is frequently sub-periosteal—green-stick fracture. The displacement is limited to a slight angular kink, or may be absent entirely; while the power of movement may suffer no definite interference, and the arm may be raised vertically without any hesitation. The careful observer will, however, notice that the child takes care of his injured side when playing, and that he does not willingly allow himself to be led by the affected arm. But if the child has not been carefully observed, as is so often the case, the doctor is not consulted by the parents until the thickening, due to callus, has made its appearance.

In every case of fractured clavicle our examination must include a glance at the blood-supply and the innervation of the arm concerned. Although damage to the blood-vessels and nerve plexuses are rare in connection with these fractures, they must not be overlooked when they do occur.

(2) Injuries in the vicinity of the **sterno-clavicular joint** are equally easy to diagnose. These are, with few exceptions, dislocations, and are caused by dragging on the shoulder-girdle or pressure thereon. Inspection and comparison with the opposite side indicate without any difficulty whether the dislocation is forwards, as is most usual, or upwards. Dislocation of the sterno-clavicular joint is often accompanied by other injuries, especially fracture of several ribs, when there has been severe compression of the thorax. The dislocation may easily be overlooked in such cases, because the fractured ribs and the contusion of the lung, which nearly always complicates these severe injuries, concentrate all attention.

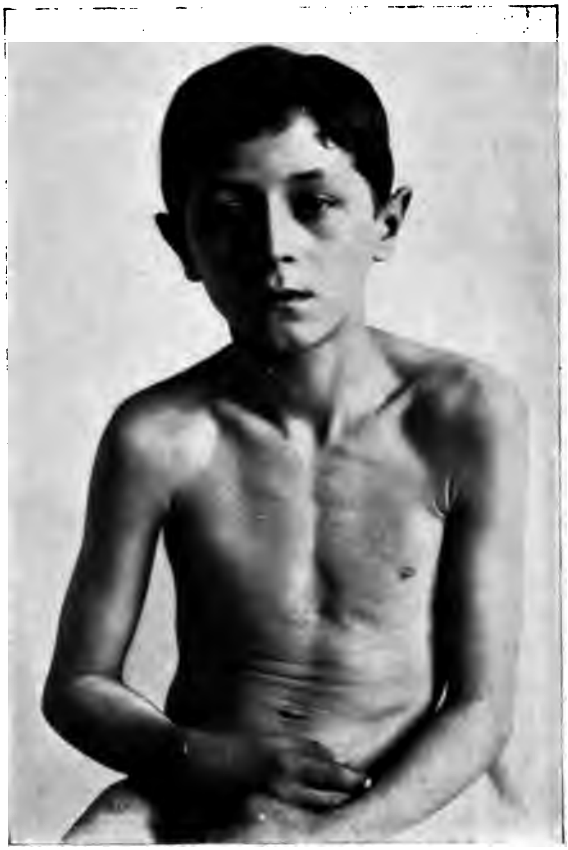


FIG. 313.—Fracture of the right clavicle.

Dislocation of the clavicle behind the sternum is somewhat difficult to diagnose and may easily be overlooked when the injury is recent, because the effusion of blood obliterates the step-like gap

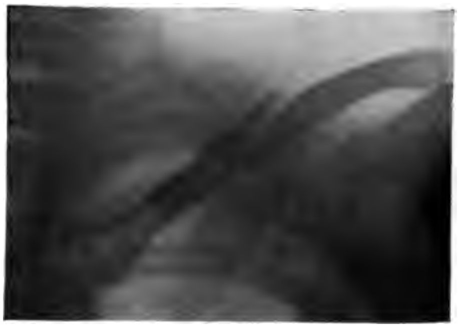


FIG. 314.—Fractured clavicle.

between the clavicle and sternum. But if the possibility of the injury is kept in mind, the diagnosis can be made by comparing the measurements, on either side, from the acromion to the sternum. Palpation of the neck will show that it is filled up by the dislocated clavicle. The diagnosis will be confirmed by a skiagram, taken with the plate on the sternum.

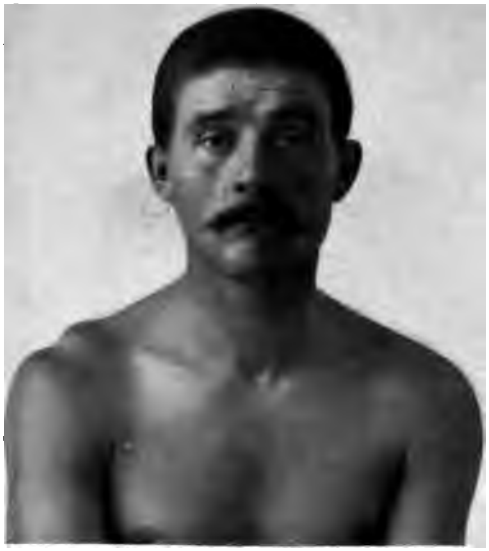


FIG. 315. -Right acromio-clavicular dislocation.

(3) **Injuries in the neighbourhood of the acromio-clavicular joint** are more interesting from the diagnostic point of view, because they are more frequent. The differential diagnosis concerns contusion, sprain, and dislocation of the joint, fracture of the acromion, and of the extremity of the clavicle. We differentiate the following possibilities.

(*a*) If there be no deformity, and pain is the only symptom, we have to distinguish between *contusion* and *sprain*. We may only assume the former when the joint has sustained a *direct* injury. We will probably find some evidence of this in bruising which has supervened, or even in some abrasions. If the injury was *indirect*, the case

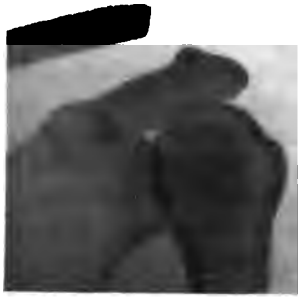


FIG. 316. -Skiagram of fig. 315 (taken from behind, and therefore, apparently reversed).

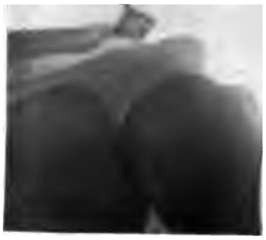


FIG. 317. -Detachment of distal end of clavicle.

can only be one of sprain. If the pain is mainly a pressure pain, affecting the whole joint, it strongly suggests contusion, whereas, if it is elicited mainly on movement—raising the arm beyond the horizontal—and if the pressure pain is limited to the fold of the joint, it suggests sprain.

(*b*) A slight deformity, consisting of a little step-like ascent from the acromion to the clavicle, may be regarded as a *sprain*—although the condition is one of loosening of the ligaments which approximates very closely to a luxation. There is no sharp line of demarcation between a sprain and a subluxation. On the contrary, fractures of the end of the clavicle or acromion may be mistaken for sprains or contusions if they are periosteal, and therefore produce no dislocation. If there is also no crepitus on movement, nothing but a skiagram can demonstrate the presence of the fracture.

(*c*) If, however, we do find definite deformity—and this usually consists of a step-like drop of the shoulder (fig. 315), we must distinguish between (1) *fracture of the outer end of the clavicle;* (2) *acromio-clavicular dislocation;* and (3) fracture of the *acromion.* Some indication is afforded by the position of the maximum pain on pressure and of the displacement. The palpation of both sides and comparative measurements will usually clear up the condition. In doubtful cases the irregular shape of the prominent edge and the crepitus permit of the distinction between fracture and dislocation. The differential diagnosis has usually to be made between dislocation and fracture at the end of the clavicle. In addition to the results of palpation and measurement, we must also take into consideration the visible external deformity. A very striking step-like formation suggests dislocation rather than fracture. The severity of the pain on pressure is also of some value, because it is more pronounced in fracture than in dislocation. The skiagrams are easy to interpret, and are conclusive (figs. 316 and 317).

There may be some difficulty in interpretation in the case of children, because the lateral cartilaginous portion of the clavicle is transparent. A fracture of the cartilage may therefore be mistaken for a dislocation. This error may be avoided by controlling the skiagram by a comparative measurement of the median fragment.

It may be noted finally that the distal end of the clavicle has occasionally been displaced *under the acromion,* and even *under the coracoid process.* These rare injuries are quite recognizable by those who are skilled in palpation.

CHAPTER LXXXII.

FRACTURES AND DISLOCATIONS ABOUT THE SHOULDER-JOINT.

A MERE glance and an examination of the power of movement usually suffice to make a diagnosis in the case of a patient who has just fallen upon his shoulder, or upon his arm, the latter outstretched to avert the fall. If we employ palpation in addition, it is hardly possible to err. The fact that so many errors of diagnosis are actually made is due to a want of anatomical consideration. A skiagram, which we may adopt as our last resource, ought only to confirm an already made diagnosis and reveal a few details, but should never be a substitute for diagnostic reflection.

We place the patient before us, with the upper part of his body undressed, and direct him to slowly lift both arms from the side. If the injured arm can be raised perpendicularly, the shoulder-joint is free, and there is certainly no serious injury of the shoulder or shoulder-girdle. In the case of sub-periosteal fracture of the clavicle in children, free movement may still persist, as stated in the previous chapter.

If the arm on the injured side can only be raised above the horizontal line in a hesitating manner, we should let it down again gradually. If the patient lets it drop from the horizontal position with a sudden grimace of pain, we may be almost positive that the collar-bone is broken.

If the arm cannot be raised at all, or only very slightly, or if the patient supports it with his other hand, it indicates the presence of a severe injury—a dislocation or fracture about the shoulder-joint.

In dislocations it may be possible, in some circumstances, to raise the arm as far as the horizontal already on the second day, with a certain amount of pain. In these cases, however, it will be observed that the movement chiefly takes place at the clavicular joint.

If the axis of the humerus is deviated inwards so that the continuation of its line would intersect the clavicle, and if the elbow is abducted from the side of the body, the case is either one of dislocation or of fracture. If the curve of the shoulder is flattened (fig. 318) it is a dislocation; if the curve is retained, it is a fracture (fig. 320). This flattening, may, however, be concealed by an extravasation of blood (fig. 319); but if dislocation be present in such a case, pressure with the finger below the acromion would show that the glenoid fossa is empty, in comparison with the other side. This sensation of the emptiness of the glenoid fossa is so unequivcoal that if it is not present, we may positively exclude dislocation in any doubtful case. Unless a hæmatoma had attained an extraordinary size, it could not prevent the detection of the gap in the normal position of the head of the humerus. Should any doubt still remain, we must try passive movements. If these are limited in certain directions, the case is certainly one of dislocation; if they are normal or unusually free, especially in the direction of adduction and outward rotation, the case is one of fracture. If the glenoid cavity is empty, but crepitus and preternatural mobility are present, the rare combination of fracture with dislocation exists.

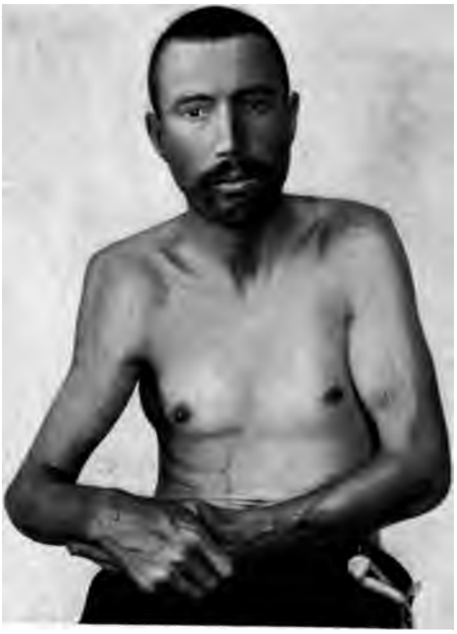


FIG. 318.—Sub-coracoid dislocation of humerus. Arm abducted. Axis deviated inwards. Shoulder flattened.

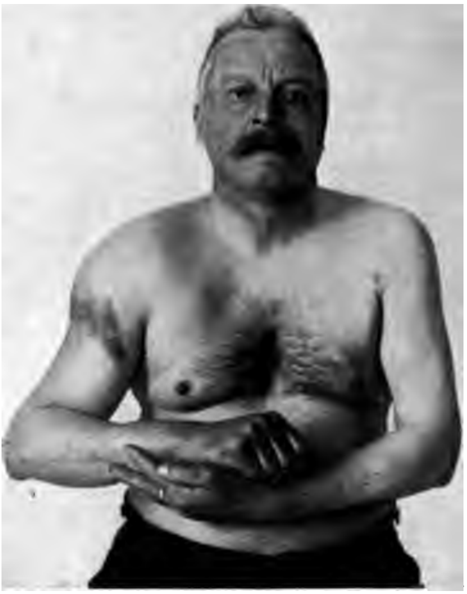


FIG. 319.—Axillary dislocation of humerus. Arm greatly abducted. Axis deviated inwards more than in adjoining case. Shoulder bulged by a large hæmatoma.

A.—DISLOCATIONS.

Having diagnosed a **dislocation**, we have next to decide whether it is of the axillary (sub-glenoid) or sub-coracoid variety: other forms are exceedingly rare. If the arm is greatly abducted from the side (fig. 319) and the head of the humerus is distinctly felt in the axilla, the dislocation is axillary. If the arm is less greatly abducted and the head can be felt and seen under the coracid process (fig. 318), the dislocation is sub-coracoid. The fact that the head of the bone can be seen and felt in Mohrenheim's fossa must obviously dispel any doubt about the presence of a dislocation.

We purposely do not lay too much stress upon the demonstration of the head of the humerus in a misplaced position, because a dislocation can, and should be, recognized without this sign. When there has been much extravasation of blood with infiltration of the soft parts, especially in an axillary dislocation, it is often impossible to make this examination without an anæsthetic, and many a dislocation would be overlooked if the diagnosis depended upon the discovery of the head of the humerus in an abnormal position.

In the absence of any considerable extravasation of blood, an indication for distinguishing between the two forms of dislocation may be obtained from the amount of *increase in the circumference of the shoulder-joint*, as measured through the axilla and over the acromion. If the increase does not exceed 2 cm. the dislocation is sub-coracoid, but if it is as much as 4 cm. the dislocation is axillary. In these cases, however, palpation is quite easy, and if there is much extravasation of blood the sign is of doubtful value.

The apparent lengthening of the arm in cases of sub-coracoid dislocation, is a point worth mentioning. This is particularly noticeable when the elbow is extended.

One who is able to recognize these two important varieties of dislocation of the shoulder will have no difficulty in detecting the rare forms, wherein the head of the humerus is *in front, under the clavicle* (subclavicular dislocation) or *above the coracoid process* (supra-coracoid dislocation) or *upwards and backwards* (sub-acromial dislocation) or *backwards and downwards* (infra-spinous dislocation). We must always be guided by the three above-mentioned cardinal signs, (1) absence of the head of the humerus from its normal position, (2) diminished range of passive movements, and (3) the presence of the head in an abnormal position, a sign which can, as a rule, be easily demonstrated in the forms last noted.

B.—FRACTURES.

If the head of the humerus is in its normal position, and passive mobility is increased, or remains normal, while active movements are quite impossible, and the power of the shoulder is lost we diagnose a **fracture.** This may be confirmed by feeling, or even by hearing *crepitus* on manipulation. This symptom is, of course, conclusive when present, but its absence is not in any sense an argument *against* fracture. The lower fragment may be so much displaced that it no longer rubs up against the upper one, or the crepitus may fail owing to impaction of the fragments, or to the sub-periosteal position of the fracture. In cases of separation of the epiphysis in young people, the crepitus is nothing more than a soft grating which may easily be overlooked.

Displacement of the lower fragment often furnishes further confirmation of the diagnosis of fracture. This fragment may be in a position of abduction or of adduction, and may also be completely displaced, either backwards or forwards. We have already studied the *position of abduction*, in which the elbow projects away from the side and in which the axis of the humerus is directed towards the centre of the clavicle (figs. 320 and 324), because of its similarity with the usual posture of the arm in dislocation. The *position of adduction* is indistinguishable from the normal posture of the arm, and therefore can only be recognized on a skiagram (fig. 326). The *forward* displacement of the shaft of the humerus is of greater importance. This displacement is recognized by the fact that the axis of the arm, when looked at from the side, does not go through the acromion, but in *front* of it. If there is any doubt about this, we can always feel that the margin of the lower fragment presses, as a sharp edge, against the anterior surface of the shoulder circumference; indeed the skin itself may be impaled by the lower fragment, so that a dimple forms.

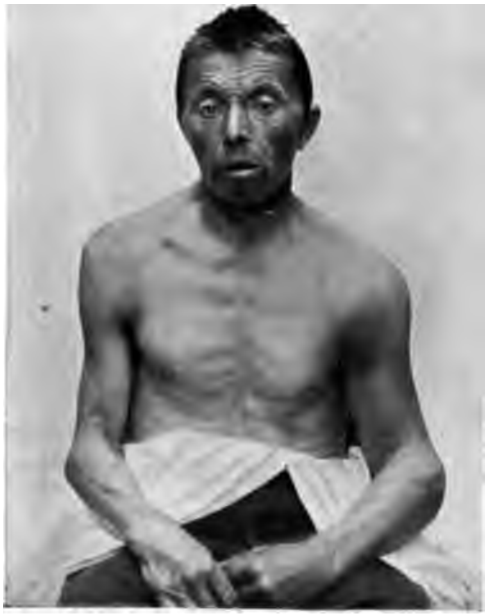


Fig. 320. — Fracture through the tuberosities. Arm abducted, but curve of shoulder maintained (after Kocher).

In some cases nothing abnormal beyond a diffuse swelling is visible on inspection, and neither unnatural mobility, nor crepitus, can be detected, owing to the impaction of the fragments. Comparative measurements of the length of the humerus from the acromion to the external epicondyle may also fail to give any positive indication. If it were not that the persistent loss of power pointed to some severe injury, one would be inclined to be content with the diagnosis of contusion of the shoulder. In such cases we are assisted by the presence or absence of "fracture-pain." We press, with equal force, the tips of two or three fingers under each acromion. A circumscribed severe pain resulting from this pressure points to fracture. Then we test whether axial pressure elicits pain ; this is done by pressing the elbow upwards, with counter-pressure over the shoulder. In recent cases, the presence or absence of pain on axial pressure is decisive, for or against fracture respectively.

If there is no displacement, this pain on axial pressure may be very indefinite, and disappear after twenty-four hours, although pain on local pressure continues.

It is never justifiable to attempt any extensive movements under an anæsthetic when the displacement is slight or altogether absent. Such may result in causing a very undesirable amount of displacement. The diagnosis of fracture can be based upon the complete loss of power and the pain on pressure. The variety of the fracture can be ascertained by direct palpation, and by noting whether the great tuberosity does or does not follow the movement of the bone on rotating it.

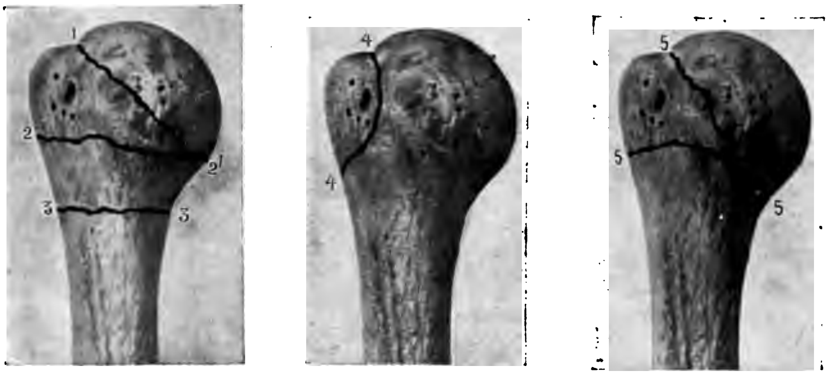


FIG. 321.—The most frequent varieties of fracture at the upper end of the humerus.

1. Fracture of the anatomical neck.
2. Fracture through the tuberosities.
3. Fracture below the tuberosities.
4. Fracture of the great tuberosity.
5. Y-Fracture.

We are now in a position to diagnose the precise form of fracture, if we take into consideration the typical lines of fracture shown in fig. 321.

It is only possible actually to feel the fracture when it is extracapsular. Therefore if the edge of a fragment is felt the fracture must either be **through the tuberosities,** or **below the tuberosities.** The distance of the edge of the fragment from the joint indicates which of these two varieties it is. If the edge cannot be felt, one must test whether the great tuberosity follows the movements of the bone. If the great tuberosity moves with the humerus on rotation, the fracture must be intrascapular—probably a **fracture of the anatomical neck,** assuming that our diagnosis of fracture is confirmed by crepitus. But if we have not elicited any crepitus, and have only based our diagnosis on the presence of pain on axial pressure, there may be an impacted fracture between the tuberosities which is the more frequent event.

But if the great tuberosity does not move when the humerus is rotated, the choice lies between the fracture which goes through the tuberosities, and the fracture which runs below them. Even if we cannot feel the edge of a fragment, the diagnosis can be made with considerable accuracy from the position of the maximum pain on pressure.

Fracture of the metaphysis caused by a wrench produces the least symptoms. We will describe this form of fracture in connection with the radius, but Iselin has shown that this variety also occurs at the upper part of the diaphysis of the humerus. The diagnosis can only be made from the local pain on pressure.

If the **great tuberosity is detached as an isolated fragment,** theoretically it should not move when the humerus is rotated. This injury may be the result of mere muscular contraction, but is usually associated with a dislocation. There is, however, so much effusion of blood and muscular tension, that very little can be made out by palpation, unless an anæsthetic is given. It is quite the usual thing to overlook a detachment of the tuberosity in cases of dislocated shoulder. But whenever there is a circumscribed severe tenderness in the region of the great tuberosity, and the bone seems to be widened, this possibility should be thought of and a skiagram taken. As a point in diagnosis, it should be remembered that external rotation of the arm is impossible in this injury, because the external rotators (supra- and infra-spinatus, teres minor) are inserted into the great tuberosity, and the arm can, at most, be raised only as high as the horizontal.

Special mention should be made of *separation of the epiphysis,* in infants during delivery, and *separation of the epiphysis in infants with congenital syphilis,* owing to syphilitic osteo-chondritis. The loss of movement in the arm is the most striking symptom in these children—so-called "pseudo-paralysis."

If the shoulder is flattened and the head of the humerus is approximated to the thorax, just as in the case of dislocation, but if, at the same time, there are increased mobility, crepitus and pain on axial pressure as in a fracture, though nothing can be found wrong in the humerus, we should think of a **fracture of the neck of the scapula,** more especially of detachment of the articular process and coracoid process. Confirmation of this diagnosis is furnished if the deformity is corrected by displacing the humerus to the side and pressing upwards, and by the return of the deformity immediately the pressure is relaxed.

Figs. 322-329 indicate the skiagraphic appearances. The importance of taking a lateral view (if possible with the arm abducted) is shown by comparing figs. 328 and 329.

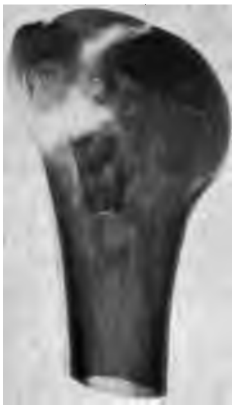


FIG. 322.—Shattering of head of humerus (patient was run over by railway carriage).

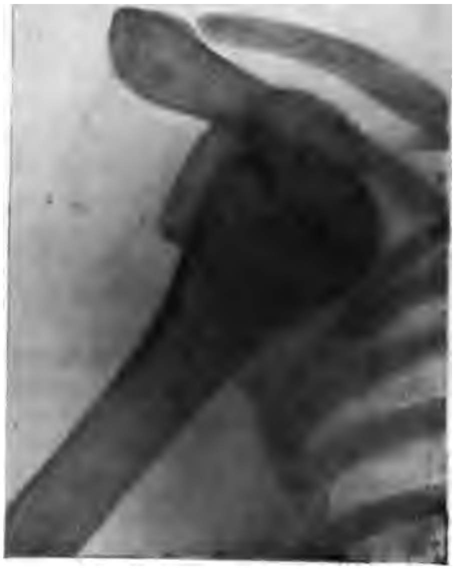


FIG. 323.—Sub-coracoid dislocation.

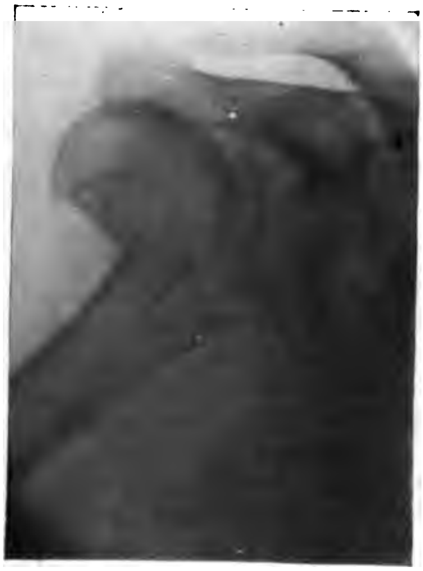


FIG. 324.—Fracture of humerus, through tuberosities, in position of abduction, as in dislocation (*cf.* fig. 320).

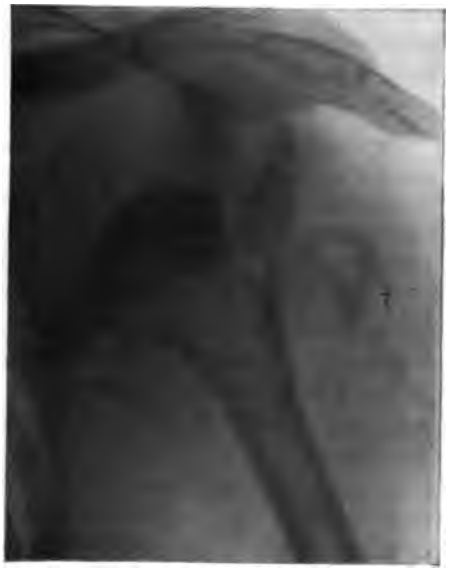


FIG. 325.—Axillary dislocation with detachment of great tuberosity.

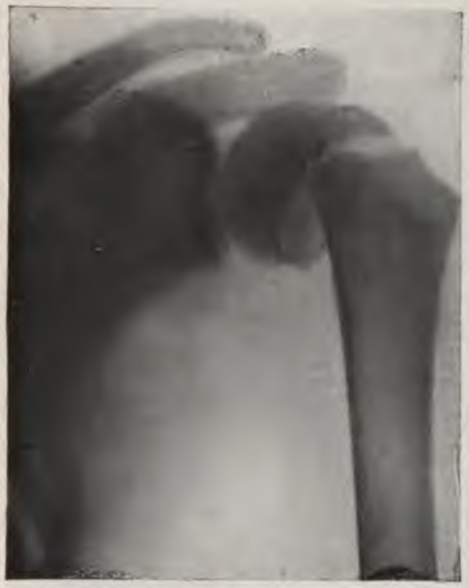


FIG. 326.—Fracture of humerus through tuberosities in position of adduction.

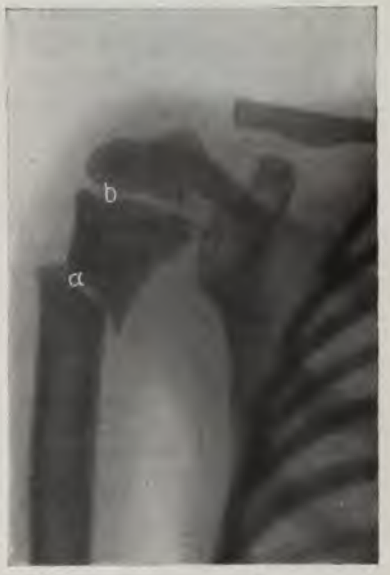


FIG. 327.—Fracture of humerus, below tuberosities: (a) line of fracture; (b) epiphyseal line.

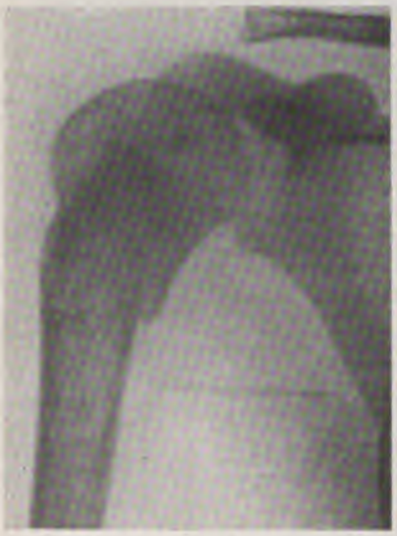


FIG. 328.—Fracture below tuberosities. Taken from the front. Displacement apparently slight.

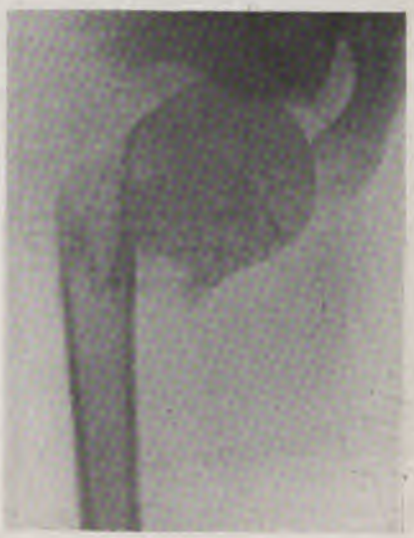


FIG. 329.—Same case, taken from the axilla. (Tube in axilla, plate on shoulder.) Considerable displacement obvious.

Forward displacement of the lower fragment occurs much more frequently than one realizes.

We should only be content with the diagnosis of **contusion** or **sprain**, if nothing is yielded by an examination, carried out as here directed, whether the injury be direct or indirect. We append to this chapter a summarized table which will facilitate diagnosis in any given case.

Curve of shoulder flattened	Glenoid fossa can be deeply pressed into; head of humerus to be felt in an abnormal position.	Passive movements limited in certain directions (adduction).	(1) **Dislocation of humerus** (usually axillary or sub-coracoid).
		Passive movements free.	(2) **Dislocation with fracture.**
	Glenoid fossa cannot be deeply pressed into; head of humerus not felt in an abnormal position.	Head of humerus shows nowhere circumscribed pain on pressure (not even through the axilla), but the scapula is painful on pressure through the axilla. Coracoid process follows the movements of the arm. The whole shoulder region can be displaced upwards, but immediately sinks downwards again.	(3) **Fracture of neck of scapula.**
		Circumscribed pain in head of humerus on pressure.	(4) **Fracture of humerus with position of abduction.**
Shoulder curve retained.	Curve over shoulder can be deeply pressed in; passive movements limited; head of humerus can be felt in abnormal position.	.. ..	(5) **Dislocation of humerus with hæmatoma.**
	Curve over shoulder cannot be pressed in; passive movements free; head of humerus not felt in abnormal position.	No pain on axial pressure; loss of power moderate; no local pain in bone on pressure; pain about the capsule.	(6) **Sprain of humerus.**
		No pain on axial pressure. Severe circumscribed pain on pressure over great tuberosity; active outward rotation, *nil*. Rarely tuberosity can be felt to be movable.	(7) **Fracture of great tuberosity.**
		Pain on pulling or pressure in long axis; local pain on pressure, especially through axilla; great tuberosity follows rotation; severe loss of power.	(8) **Fracture of head or anatomical neck.**
		Ditto, but the pain on pressure through axilla is felt in scapula and not in head of humerus (see also above).	(9) **Fracture of neck of scapula.**
		Ditto, but pain on pressure at level of tuberosity. also demonstrable from outer side; loss of power slight or disappears rapidly.	(10) **Impacted fracture through tuberosities.**
		Ditto, but great tuberosity does not follow rotation; lower fragment often displaced anteriorly; anterior margin can be felt through deltoid; loss of power more than in 10, but in children may be slight.	(11) **Free fracture through tuberosities** (separation of epiphysis) either without displacement or in a position of abduction, or displacement anterior.
		Ditto, but local pain on pressure below the tuberosities; site of fracture clearly felt from the axilla; loss of power very great.	(12) **Fracture of humerus below tuberosities** (surgical neck).

CHAPTER LXXXIII.

INFLAMMATORY PROCESSES ABOUT THE SHOULDER-JOINT.

INFLAMMATION about the shoulder is situated, apart from rare exceptions, in the *bursæ*, *the joint*, or in *the bones*.

A.—DISTINCTION BETWEEN BURSAL AND JOINT DISEASE.

The *subscapular bursa* and the *bursa between the tuberosities* are only dilatations of the capsule of the joint, and require no special attention, because they are never diseased independently. The *sub-coracoid bursa* is too small to be of any importance. The *bursa under the deltoid* is, however, of importance pathologically. It lies between the deltoid muscle, the capsule of the joint and the humerus, and is often subdivided into two parts, an upper, the *sub-acromial bursa*, and a lower, the *sub-deltoid bursa proper*.

How can we distinguish between an effusion into this bursa and an effusion into the joint?

In the first place by *inspection*. If the sub-deltoid bursa is filled up, it raises the deltoid muscle from off the joint and the humerus, especially at its external and anterior portions. Küster has pointed out that this bulging is best recognized by looking at the patient from behind and above, and comparing the two shoulders. On the other hand, an intra-articular effusion does not make the curve of the shoulder any more pronounced, because even a tensely-filled capsule is unable, for anatomical reasons, to lift up the deltoid to any considerable extent. If the deltoid muscle bulges to a great distance downwards, an intra-articular effusion is at once excluded, and we must assume that the effusion is in the deltoid bursa. If the effusion in the joint is so great that it causes a visible swelling, this swelling will also manifest itself where the joint is least covered by muscles, i.e., in the posterior region, and also in the diverticulum of the capsule, along the biceps tendon.

This does not imply that the shoulder-joint which contains an effusion does not appear to project away from the body more than on the healthy side. But this is due to the fact that an extensive effusion presses the humerus and all that covers it somewhat away from the body. This broadening of the shoulder, as seen from the front is, however, quite different from the protuberant bulging just described in connection with effusion into the bursa. The outline of the shoulder is otherwise retained.

If, on palpation, it appears that it is something like a tense cushion under the deltoid and over the bone which causes the enlargement of the shoulder, the case must be one of a bursal effusion. But if we at once come upon the humerus under a muscle of normal thickness, and the shoulder is nevertheless widened, as seen from the front, the humerus must be displaced by an articular effusion.

A *bursitis* is only painful on pressure over the area of the bursa, but an arthritis is painful over the whole extent of the joint, especially if pressure is made directly over the capsule, i.e., at its posterior and inferior regions.

Loss of power is another piece of evidence in favour of disease of the joint.

In disease of the shoulder-joint, as in that of other joints, there is always the reflex attempt to keep the joint at rest, and allow its function, as far as possible, to devolve upon some other joint. In the hip this "muscular fixation" is at once manifest as a limp, because the joints of the lumbar vertebræ only make a poor substitute for it. But in the shoulder, the joints at the two extremities of the clavicle are able to compensate for it, to a considerable extent, and thus conceal the loss of movement. We must, therefore, examine for loss of power, bearing in mind the behaviour of the scapula and clavicle. In the ordinary way, the scapula does not participate in the movement of the shoulder until the arm is raised above the horizontal. But if the scapula participates before the arm reaches this level, it is obvious that there must be some limitation of movement in the shoulder-joint. A vertical position of the clavicle and the approximation of the shoulder to the middle line, also signify limitation of movement. Fig. 330 (left-sided muscular fixation of the shoulder-joint) illustrates this latter symptom very clearly. If this stiffness is only occasional, e.g., when the patient is tired, or if it disappears under anæsthesia, it is purely of a muscular nature. If some force is required under the anæsthetic to overcome the stiffness, it indicates that definite changes have already occurred, especially contraction of the capsule and adhesions between the head of the humerus and the glenoid fossa. If the joint remains entirely fixed, even under the anæsthetic, there are either very old-standing fibrous adhesions, or bony ankylosis exists.

The patient chiefly notices this disability, whether it be due to the muscle or fibrous tissue, in the movements wherein the rest of the shoulder girdle cannot act as a substitute—e.g., in adduction of the arm to the middle line behind the back. Interference with this movement is sometimes the first complaint in tubercle of the shoulder-joint.

There are three conditions which we must guard against, in examining the function of the shoulder-joint. The first has been included under the general term of "*joint-neurosis*," and usually occurs in the form of *hysteria*.

A "neurosis of a joint" may occur in children, especially in little girls. If the patient has sustained an injury and looks forward to compensation, it is dignified with the designation of "traumatic neurosis." We cannot investigate here the respective parts played in this condition by deliberate exaggeration and by involuntary auto-suggestion. These, no doubt, vary in different cases. But the fact is that many patients who assert that they can hardly lift their arm to the horizontal are often able, within a few minutes, to raise it quite vertically, provided that the neurotic habit is not too deeply rooted within them. As a rule there is no interference with passive movements, and this, of course, excludes the possibility of any serious injury to the joint. But we must be careful not to fall into the second source of error, viz., a *genuine paralysis*.

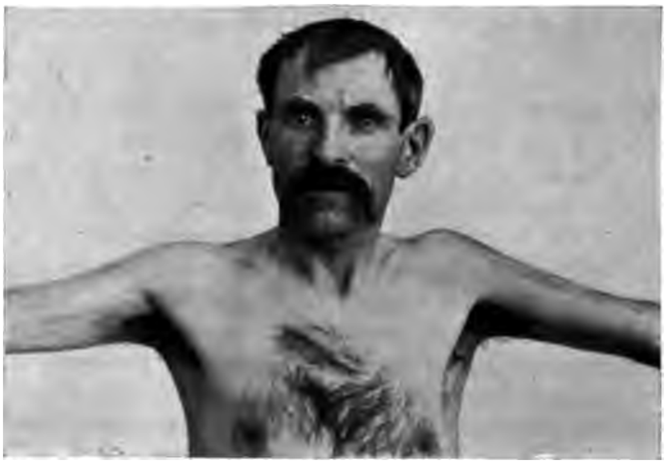


FIG. 330.—Approximation of the level of the shoulder to the middle line, and steep position of the clavicle, when the arms are raised, signs of stiffness of the shoulder (early stage of arthritis of shoulder).

A workman dislocates his shoulder. It is promptly reduced by a doctor, but still remains powerless; the patient cannot raise his arm. At first the doctor thinks of some damage to the joint through the injury. Then he thinks that the patient may be malingering, because the passive movements are quite free. Examination, however, shows that the deltoid does not contract at all when the movements are being tested, and that the reaction of degeneration is present. The patient has therefore, sustained a *paralysis of the circumflex nerve*, as a result of his dislocation.

Malingerers and hysterical patients usually contract their muscle somewhat, in order to show their "good intentions"; whereas in paralysis this is impossible. But *paresis* may cause great difficulty, because a certain amount of voluntary contraction occurs, just as in malingering. We can generally escape from this difficulty, however, by examining with the faradic and galvanic currents, and by testing the sensation in the area supplied by the circumflex nerve—i.e., over the deltoid muscle.

If we base our differential diagnosis between bursitis and arthritis of the shoulder-joint on what has already been said, it will follow that the only movement which is hindered in bursitis is the one wherein the inflamed bursa is compressed between the acromion and the humerus—i.e., raising the arm from the side; the other movements are quite free. But when the joint itself is inflamed and its function is interfered with, all the movements are more or less limited, both active and passive.

B.—DIAGNOSIS OF THE VARIOUS FORMS OF BURSITIS AND ARTHRITIS OF THE SHOULDER.

(1) BURSITIS.

In rare instances acute infections, like gonorrhœa, acute articular rheumatism and staphylococcic infections (furuncle), &c., may give rise to bursitis by metastasis. The most frequent forms are, however, the **traumatic** and the **tubercular.** If the disease appears directly after a contusion of the shoulder, or after the constant repetition of a slight trauma—including any unaccustomed work which involves the deltoid muscle—we may assume that it has a traumatic origin. But if there has been a considerable free interval between the injury and the onset of the bursitis, or if the disease has come on gradually and quite spontaneously, we must think of tubercle, especially if this is supported by a hereditary predisposition, and by the previous history of the patient.

Occasionally the bursitis is not primary, but has been caused by the rupture into the bursa of an extra-articular focus in the bone. Positive information can only be furnished by a skiagram.

(2) ARTHRITIS OF THE SHOULDER.

In discussing the etiology of this condition, it is necessary to distinguish the acute from the chronic cases.

(*a*) *Acute inflammation of the shoulder-joint* may be one symptom of **acute articular rheumatism.** Such a case would be left to the physician, but we must remember that there is a complete chain of transitional forms—from the purely serous polyarthritis of rheumatism to the suppurative inflammation of pyæmia—which, under certain circumstances, may require surgical treatment. The most important of these is **scarlatinal arthritis,** which often attacks the shoulder; and we must be careful lest we compromise the functions of the joint by delaying incision too long. **Gonorrhœal arthritis** is another transitional form between rheumatism and pyæmia, and is usually recognized by the fact that only one joint is affected. This variety should be thought of, if the patient has outgrown the stage of children's ailments, but has not yet learnt how to avoid the gonococcus. But one must never forget that this micro-organism may occur in childhood (especially in little girls)—*Gonococcus insontium.* **Pyæmic arthritis of the shoulder** is mostly met with in puerperal infections, but may occur in any pyæmic disease, especially septic endocarditis.

(*b*) The diagnosis is much more difficult in *chronic arthritis of the shoulder.* It is most important, from the therapeutic point of view, to decide whether it is **tubercular** or not.

In the first place one has to consider **traumatic arthritis of the shoulder.** The histories of these cases are very similar, with slight variations. A middle-aged or elderly man has sustained a sprain of the shoulder, or some severe injury, such as a dislocation, which has been properly reduced. The patient may, of course, be a female, but the condition rarely happens in this sex. The original pain disappears quite normally, but there is no restoration of the power of the joint. Attempts at movement remain painful, and they are often attended by creaking and grating. Pain frequently radiates towards the back of the neck and the elbow. On examination, it will be found that the joint is more or less stiff, and that the capsule is distinctly painful on pressure; but there is not sufficient effusion to permit of recognition. If untreated, the condition may persist for weeks and months, but in slight cases it rapidly yields to proper treatment, especially if the patient is otherwise well, and has not instituted a claim for damages. In old people, in rheumatic and gouty subjects, this traumatic arthritis of the shoulder may develop into one of the varieties of "chronic rheumatism," which proves refractory to all treatment.

If this disease has not followed the injury, but has come on gradually some time after the subsidence of the immediate effects of the accident, we should think of *post-traumatic tuberculosis.*

This term is justifiable if the injury has determined the localization of the tubercle, or has converted a previously existing latent tuberculosis into the active variety. The diagnosis of tubercle is based upon the long continued obstinacy of the trouble, a positive von Pirquet's test, the previous history of the patient, and a skiagram. The history of the case and the probable age of the changes revealed by the skiagram will help to decide whether there had been any antecedent disease of the joint.

The diagnosis is easier if there has been no antecedent injury, for then it is only necessary to decide between **tubercle** and **chronic articular rheumatism.**

The *anatomical* changes which occur in this latter include processes of serous effusion, fibrous adhesions, proliferation, and destruction—indeed, any morbid change which may occur in a joint. Their *etiology* includes injuries, toxic processes (lead), infections (an original acute infective rheumatism), and finally neuropathies (tabes, syringo-myelia) apart from cases which we cannot account for at all. It must be stated, however, that none of the anatomical varieties presupposes any definite etiology; one and the same cause may be responsible for the most differing anatomical forms.

The tendency towards the affection of many joints in a symmetrical manner is an important diagnostic sign, common to all varieties, whatever be their causes—even if traumatic. This circumstance permits the diagnosis to be made at once in a large number of cases. It is true that tubercle often affects many joints, but, as a rule, there is at any rate one focus so much involved that there is no difficulty about the diagnosis. It is more difficult to diagnose the cases of subacute or chronic rheumatic polyarthritis, wherein several joints are attacked at long intervals. If only *one* joint is diseased, we may remain long in doubt. Early muscular atrophy, progressive deterioration—even if slow—depression of the general health, and possibly also a slight rise in temperature, would point to tubercle; but a variable local condition with good general health would indicate a "rheumatic" affection. Some weight in making a diagnosis may also be attached to the success or failure of spa-treatment. All these difficulties apply to those frequent forms of shoulder-joint tubercle, wherein there is neither effusion nor any demonstrable swelling of the capsule, wherein the morbid process manifests itself by slow destruction and simultaneous absorption of the articular ends of the bones—a clinical picture which used to be termed "caries sicca." On the other hand, there can be no doubt about the diagnosis of tubercle if a localized swelling gradually forms, even at the posterior region of the joint, with suppurative softening of the tissues, and the formation of sinuses which discharge their pus and pieces of caseous material.

Gummatous disease of the shoulder-joint may occur, but it is so rare that it does not enter into practical consideration.

We may now briefly summarize the foregoing :—

If the signs of inflammation of the shoulder—loss of power, spontaneous pain, tenderness on pressure—come on immediately after an injury, we may assume the presence of a purely traumatic arthritis of the shoulder-joint, even if the malady persists for weeks or months. But if these symptoms come on spontaneously, or supervene a few weeks after a slight injury, we should think of tubercle, especially in young patients. If in addition to the shoulder, other joints become affected with inflammatory symptoms at longer or shorter intervals, but without, anywhere *presenting the classical signs of a tubercular focus, the case probably represents one of the forms of chronic articular rheumatism, and the older the patient is, the more probable is this diagnosis.*

C.—PRIMARY DISEASES OF THE BONE.

We have referred to the **bones** as the third source of inflammation in the neighbourhood of the shoulder-joint. So far as the bone disease manifests itself in the form of *inflammation of a joint* the foregoing remarks will apply. The involvement of the bone, or the presence of the primary focus within it can only be demonstrated by a skiagram. But the bone may be diseased, *and the joint itself not be involved.* This condition will be suggested, when there are pain and swelling in the neighbourhood of the joint, while its movements remain free. The localization of the changes will, at once, enable us to distinguish whether the disease originates in the scapula or the humerus, and its course generally enables us to determine whether it is acute osteomyelitis, tubercle or gumma. In its early stages, a *sarcoma* may be confused with a chronic inflammatory disease.

CHAPTER LXXXIV.

INJURIES ABOUT THE ELBOW-JOINT.

ALTHOUGH the elbow-joint is superficial in position and easily felt, injuries thereof are a source of great perplexity of diagnosis. There are two reasons for this; firstly, the fact that three bones participate in the construction of the joint, and secondly, the extensive swelling of the soft parts which occurs—much more extensive, for example, than in the case of the wrist. But if we accustom ourselves to draw logical conclusions from what is actually felt, we shall find that the undiagnosable injuries about the elbow will tend to become fewer and fewer.

Having decided from the loss of power and deformity that some severe injury to the elbow has occurred, we must first determine whether a fracture or a dislocation is present, and then the variety of the one or the other.

Some indications are furnished by the *age* of the patient. Fractures and separations of the epiphyses, with secondary dislocations, are more apt to occur in early childhood, whereas adults are more subject to pure dislocation, because of the relatively greater firmness of their bones.

We will now proceed to detail the methôd of examination.

I.—INSPECTION.

Sometimes a mere glance suffices for the diagnosis. If the outlines of the sigmoid fossa are visible through the skin of a thin individual and the head of the radius projects behind, no one can doubt the existence of a dislocation (fig. 331). If the axis of the forearm, as seen from the front, instead of deviating slightly externally is directed internally, we immediately think of certain forms of fracture (fig. 343), &c.

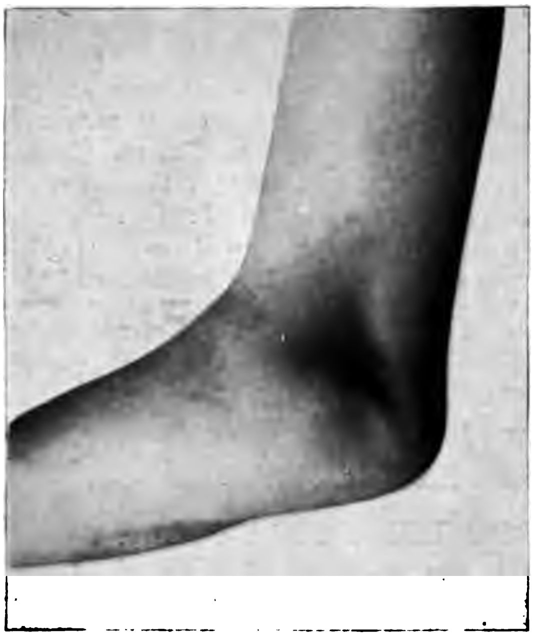


FIG. 331.—**Backward dislocation of elbow.**

On inspection we note the following points, which we shall make further use of, in the course of examination.

(1) Position of the elbow (flexion or extension, abduction or adduction, pronation or supination, abnormal position of the axis of arm or forearm). (2) Degree of the swelling. (3) Prominence of the bony parts. (4) Ecchymoses. (5) Impalement of the skin. (6) Posture of the hand (paralysis of the radial nerve).

II.—EXAMINATION OF ELBOW MOVEMENTS.

We first ask the patient to carry out a few movements in various directions. If he complies with our request, and the movements attain the normal range, he has neither a dislocation nor a fracture which interferes with the mechanism of the joint. If *active* movements are restricted, we must endeavour to ascertain the extent of the *passive* movements, at first, gently without anæsthesia, and then, if necessary, under an anæsthetic. The following possibilities have to be considered :—

(*a*) If there be an excessive amount of movement in one definite direction, while movement in the opposite direction is *restricted* by the tension of the uninjured ligaments, we may conclude that a **sprain** has occurred.

In **posterior or postero-external dislocations,** which constitute the majority of these cases, it is possible to over-extend the elbow, but it is not possible to flex it beyond a right angle. In pure **lateral dislocations** flexion is indeed possible, but the aspect of the joint, as seen from the front, with the lateral displacement of the forearm in relation to the humerus (the bayonet shape of the arm) is so striking, that the dislocation of the joint cannot be missed. We must, however, decide whether it is a complete or incomplete backward dislocation, or a dislocation backwards and outwards, or the rare backward and inward form, or the still more rare backward dislocation of the ulna only. Careful palpation of the projecting bony landmarks will furnish information on all these points.

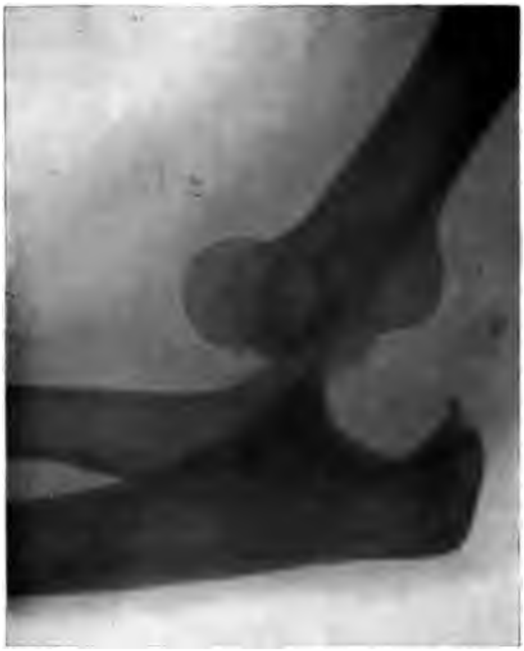


FIG. 332.—Dislocation of elbow backwards. Skiagram of fig. 331.

(*b*) If our examination has shown that there is *no interference with passive extension and flexion,* thus excluding the possibility of any ordinary dislocation, the case is one of **fracture,** or there may be no severe injury at all.

Before coming to any definite conclusion and diagnosing the exact form of fracture, it is desirable to recall the possible varieties by a glance at the accompanying diagrammatic illustration (fig. 333).

The skiagrams which follow illustrate again the most important and typical fractures, mostly of children. Owing to the epiphyseal cartilages, the interpretation of their skiagrams often presents great difficulties.

The examination of the power of the elbow and the testing for false mobility may yield the following possibilities :—

(1) *If all passive movements are free,* and the only derangement consists of the *impossibility of active extension,* it is obvious that some break has occurred in the continuity of the extension apparatus, and experience shows that this usually indicates a **fracture of the olecranon** (fig. 336).

If, anticipating the order of the examination, we palpate its surface, and come upon a gap in the bone, or find thereon a transverse, sensitive groove, or if we are able to displace the tip of the olecranon from the ulna, the diagnosis is confirmed.

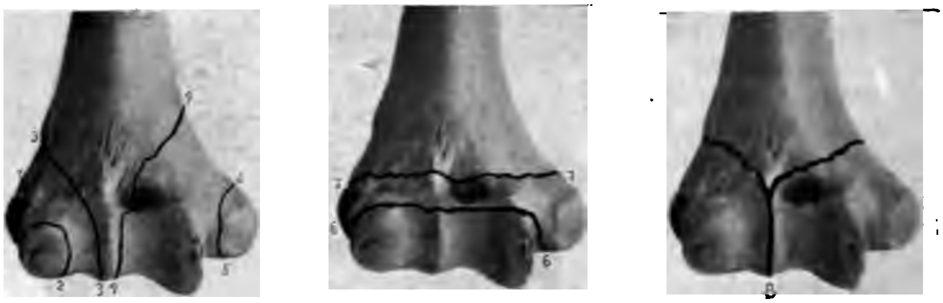


FIG. 333.—Course of the lines of fractures at the lower end of the humerus.

1. Fracture of the external epicondyle.
2. Fracture of the rotula.
3. Fracture of the external condyle.
4. Fracture of the internal condyle.
5. Fracture of the internal epicondyle.
6. Fracture through the condyles.
7. Fracture above the condyles.
8. Y-shaped fracture.

It is obvious that the examination for active extension must be conducted in such a way that the drop of the forearm by its own weight should not mislead the observer into thinking that active extension has been performed.

(2) If the olecranon is uninjured, the forearm, which is usually flexed to a right angle, should be moved backwards and forwards in relation to the humerus. If this is possible, and if the epicondyles are felt to move *with* the olecranon, there must be a break in the continuity *above* the joint, i.e., a **supra condylar fracture.**

If, on displacing the olecranon backwards, it gets into the position of a posterior dislocation, but the epicondyles do not follow it, and if it can easily be replaced into its normal position, we should think of a spontaneously reduced dislocation and fracture of the **coronoid process of the ulna,** especially if these movements are associated with some crepitus.

(3) If we cannot move the forearm backwards and forwards in relation to the humerus, we must examine for another important sign, i.e., for abnormal lateral mobility, in the sense of *adduction or abduction.*

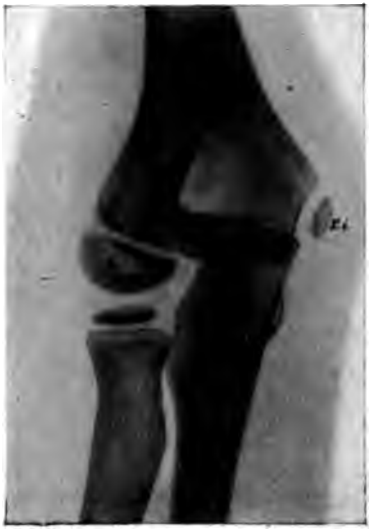


FIG. 334.—Normal elbow taken from behind. Boy aged 11. *E.i.* = centre of ossification of epicondyle. *C.e.* = external condyle. The centre of ossification of internal condyle is not yet present.

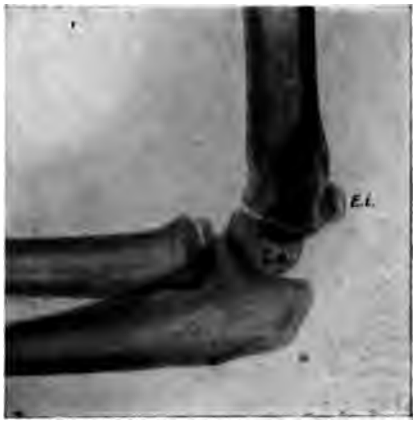


FIG. 335.—Normal elbow taken from the side. Boy aged 11. References as in previous figure.

As there is always a certain amount of abduction and adduction normally possible in children, it is necessary to compare the injured side with the uninjured one. The forearm makes an obtuse angle with the arm, the angle being open outwards, thus constituting a slight degree of valgus, as in the knee. This valgus posture is more pronounced in females than in males.

If, on comparing the two sides when both arms are kept in the same attitude, it is seen that the normal abduction is lost on the injured side (fig. 343), or, indeed, replaced by a position of adduction, we can at once conclude that there is abnormal lateral mobility. Passive movements will show whether

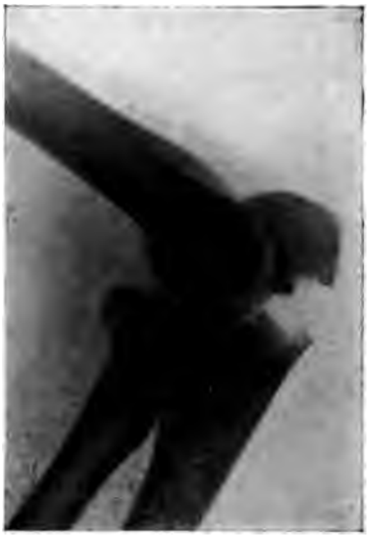


FIG. 336.—Fracture of the olecranon.

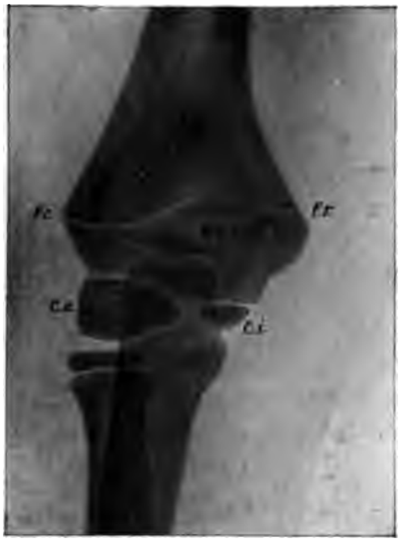


FIG. 337.—Supracondylar fracture without dislocation (*Fr.*). Boy aged 12. The internal epicondyle has fused with the humerus (unusually early). The centres of ossification for the internal condyle (*Ci.*), and the olecranon (*Ol.*) have appeared.

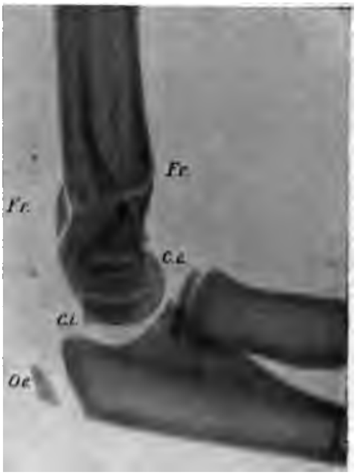


FIG. 338.—Same case as previous figure, from the side. *Fr.* = line of fracture with small piece separately broken off at the back.

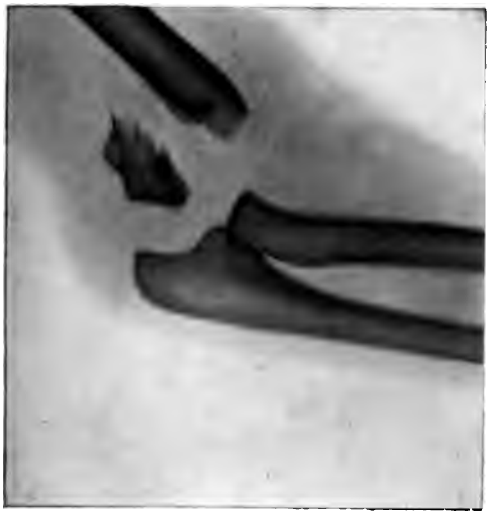


FIG. 339.—Supracondylar fracture (hyperextension fracture) in a girl aged 3, taken from the side, showing the backward displacement.

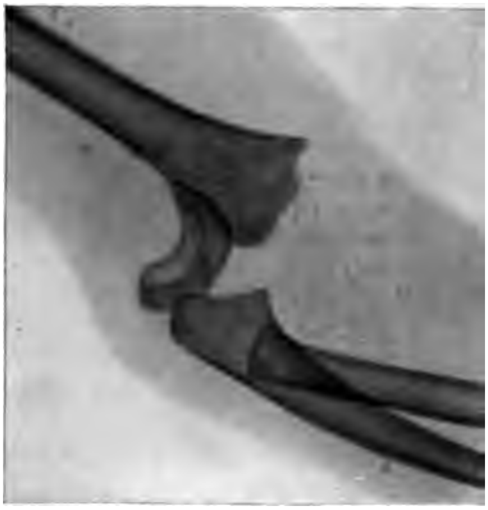


FIG. 340.—Same case, taken from the side, showing lateral displacement (bayonet shape).

this increased mobility is present in both directions, or only in one direction. In the former case the condition must be one of *supracondylar* fracture, which we have already recognized by the fact that the forearm can be displaced from before backwards. In the latter case the condition must be one of damage to the ligaments of one side, not, as a rule, merely a simple rupture of a ligament, but a *fracture* of the portion of bone into which the ligament is inserted, i.e., the *internal or external condyle or the epicondyle.* If ulnar adduction is increased, the external radial lateral ligament must have given way, or the **external condyle** may have been torn off with it (figs. 348 and 349). On the other hand, increased *abduction* signifies the **tearing off of the internal epicondyle** (figs. 344-346), or the much rarer fracture of the **internal condyle.**

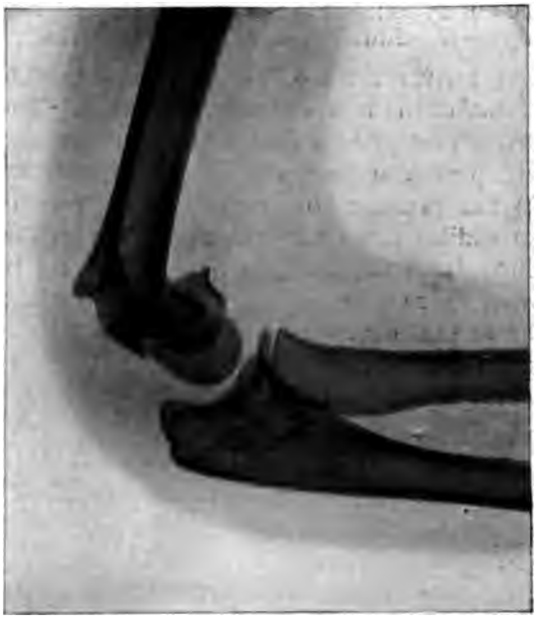


FIG. 341.—Supracondylar fracture (flexion fracture) in a boy aged 10. Lower fragment displaced to the front.

(4) But even this examination may fail to give us an unequivocal result. It may be possible to displace the forearm to some extent, both backwards and forwards, there may be increased mobility in the direction of abduction and adduction, but not one of these signs may be sufficiently pronounced to base a diagnosis thereon. We must therefore make one final test by firmly grasping the humerus at the epicondyles, and attempting to move the bones of the forearm, at the elbow joint, in a *lateral direction.* The object of this manœuvre is not to bring the forearm to an angle with the humerus, but to displace it into a direction parallel therewith. If we can do this, it is very probable that a piece of bone consisting of the trochlea and rotula has

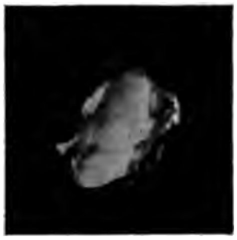


FIG. 342. — Piece of cartilage bone broken off the rotula (*cf.* fig. 350).

been broken off: in other words, that a **fracture through the condyles** has been sustained.

It is very likely that this movement may be effected in the case of other fractures which extend into the joint, especially in fractures of the condyles; but these have already been recognized by the abnormal degree of adduction or abduction of which they are capable.

(5) Even if this test is negative, we cannot definitely exclude injury of one of the bones forming the joint. There still may be a *circumscribed piece of bone broken off, within the joint*, which does not interfere with passive movements. Two symptoms will lead to this assumption: (1) the presence of crepitus when free movements are made, and (2) sudden temporary interference with these movements. We conclude from the crepitus that something is broken, and the sudden interference with movement, as occurs when a loose body is in a joint, shows that some solid substance becomes incarcerated between the ends of the bones (fig. 350). Further conclusions may be drawn from palpation.

III.—PALPATION.

This will aid us in solving the problems which have not been cleared up by inspection and by testing the movement of the joint. Palpation is easy when the case is recent and the swelling is slight; but it may be quite valueless if some time has elapsed since the accident, and the joint has become tense with effused blood and its whole neighbourhood extensively infiltrated. In these circumstances we must have recourse to anæsthesia, and massage the œdema away as far as possible, before making the examination. It is most important to determine whether the swelling is situated within the joint or outside the capsule. If the latter be the case, it is in favour of a para-articular fracture (supra-condylar).

This happened in the case illustrated in figs. 337 and 338. There was no false mobility and it was only the circumscribed swelling and pain on pressure which made one suspect a "fracture of the bone above the joint."

We have next to feel the three well-known bony landmarks, viz., the tip of the olecranon and the two epicondyles.

We know from anatomy that these three points form an equilateral triangle (fig. 347 [b] and [c] lying in the same plane as the humerus, when the arm is flexed to a right angle, whereas they are all at the same level, or, mathematically speaking, in a plane vertical to the humerus (fig. 347 [a], when the arm is extended. It is always advisable to compare the injured with the uninjured side, while making the examination, in order the better to appreciate slight changes in these relations.

The following possibilities must be taken into consideration :—

(1) If the tip of the olecranon is displaced *upwards* when the arm is extended, or displaced *backwards*, when the arm is flexed, i.e., has shifted from the plane of the humerus, whereas the epicondyles retain their relation to this bone, the case is either one of backward dislocation or fracture through the condyles, which is very rare. Marked projection of the olecranon, combined with limitation of movement—impossibility of complete flexion—points to **dislocation.**

Slight projection with free passive movement indicates **fracture through the condyles,** or **detachment of the coronoid process,** which is not quite so rare.

If the projecting olecranon, notwithstanding its backward displacement, still remains midway between the external and internal epicondyle, the case is one of **simple posterior dislocation.** If we can distinctly palpate the sigmoid fossa and the head of the radius, the dislocation is **complete ;** in other cases it is **incomplete.** If the head of the radius remains, however, in its normal position, or slightly displaced inwards, the case is an example of the rare **posterior dislocation of the ulna alone.**

(2) If the *tip of the olecranon and epicondyles are together* displaced *backwards* in regard to the plane of the shaft of the humerus, so that the relation of the epicondyles to the shaft of the humerus is altered, and so that they are movable against the humerus, the case can only be a **supracondylar fracture—a hyper-extension fracture,** whose course runs from forward and below to behind and above (fig. 339).

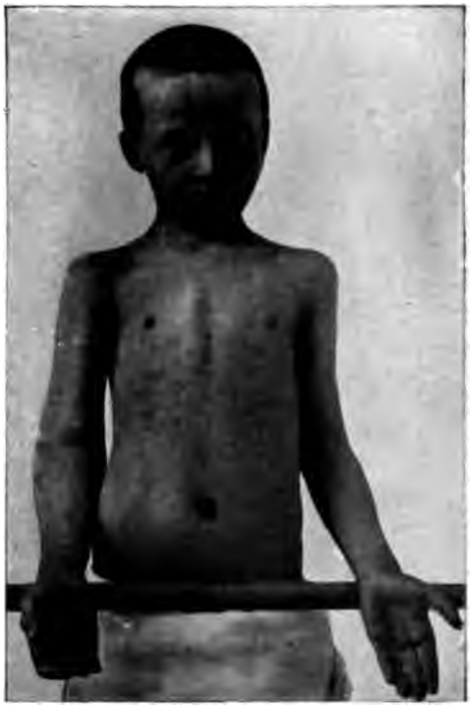


FIG. 343.—Right supracondylar fracture, in a boy aged 11. The normal cubitus valgus is straightened out (angle very definite on left side). Shortening.

(3) If the olecranon *alone,* without the epicondyles, is displaced *forwards* and movement is limited, it must be one of the rare cases of **anterior dislocation ;** if passive mobility is free, and the epicondyles

are displaced forwards at the same time, the case must be one of supracondylar fracture, arising through flexion (fig. 341). In the latter case it may be possible to feel the pointed end of the upper fragment of the humerus through the soft parts above the olecranon, if there is not too much swelling, and we may also be struck by the unusual rotundity of the elbow in profile.

(4) If the *olecranon appears to be displaced laterally* in relation to the *epicondyles*, we must note whether this displacement is in relation to *both* condyles. If it is so, there must be an incomplete or complete lateral or postero-lateral dislocation, according to the degree of ligamentous rupture and displacement.

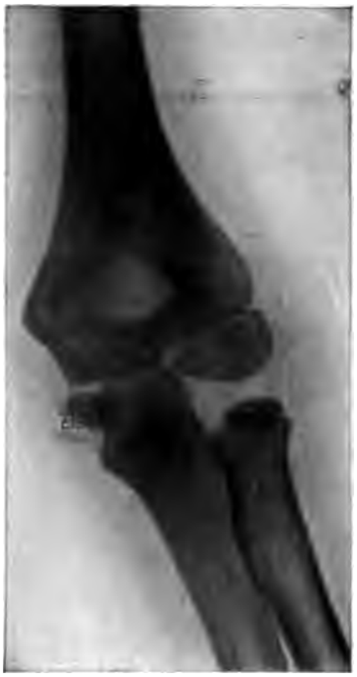


FIG. 344.—Detachment of internal epicondyle and its displacement towards the forearm.

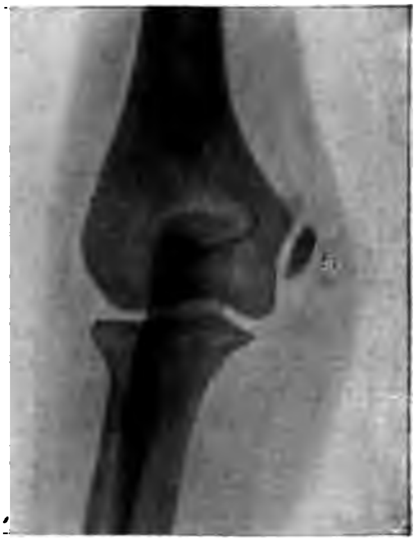


FIG. 345.—Detachment of internal epicondyle without displacement. The slight indication of callus shows, apart from the age of the patient, that it is not merely separation of epiphysial cartilage.

(5) If the tip of the olecranon only preserves its normal relation in regard to *one* epicondyle, we must assume that the other condyle or epicondyle has been broken off and displaced. The epicondyle is usually broken off by itself on the inner side, whereas on the outer side it is generally a matter of fracture of the condyle. The symptoms, in regard to false mobility, are, in principle, the same in both cases; hyperabduction when the fracture is on the inner side, and hyperadduction when the fracture is on the outer side. If the swelling

is not too great, palpation is quite conclusive. On the outer side we often find the **detached condyle** rotated to the extent of 90° or even 180°.

In fracture of the internal epicondyle the detached piece of bone is sometimes found hanging from the lateral ligament in its normal position, but often towards the anterior surface, and displaced even as far as the level of the fold of the joint (figs. 344 and 346).

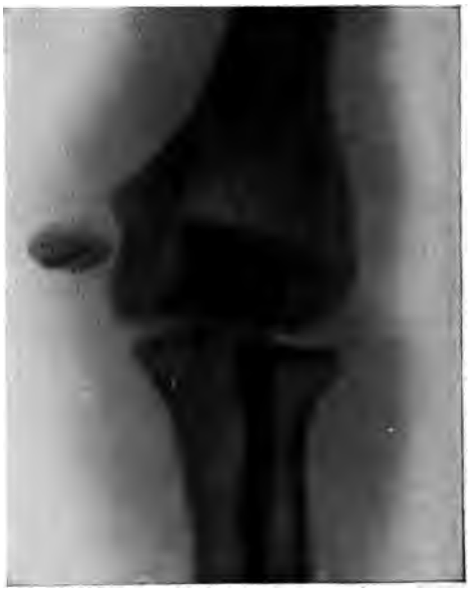


Fig. 346.—Detachment of internal epicondyle, and its rotation to the extent of 90°.

(6) If we feel the three chief points in their proper position, but the head of the radius is displaced, there must be a dislocation of the radius alone. The radius usually deviates forward (fig. 353) or outwards, rarely backwards, and the injury generally occurs in children as a result of extreme pronation combined with abduction. The so-called **subluxation of the radius** forwards, which is also frequent in children, must be distinguished from complete dislocation of the radius. This is at present looked upon as an interposition of the posterior wall of the capsule between the radius and humerus.

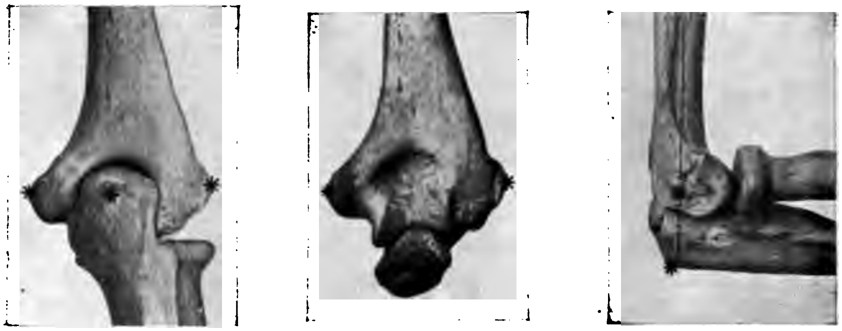


Fig 347.—Relative positions of three bony projections on elbow, which serve as landmarks.

[a] Extension. [b] Flexion as seen from behind. [c] Flexion as seen from the side.

This injury often occurs to children, when they are dragged by the arm. On palpation nothing can be found, in striking contrast to the incapability of performing any movement. The accuracy of the diagnosis is proved by the result of treatment; power of movement is at once regained if the normal conditions are restored by supination and flexion.

(7) There may be pronounced posterior displacement of the forearm, when considerable passive movement is applied to it. At the same time each separate condyle can be displaced from the shaft of the humerus. The movements are accompanied by a sound of crackling like a bag of nuts, in the joint, which is filled with blood. In these circumstances there can be no doubt that there is a supracondylar fracture, combined with fracture of the distal fragment—in other words, a T- or Y-shaped fracture (fig. 351). Fractures produced by bullets are associated with much more comminution (fig. 352).

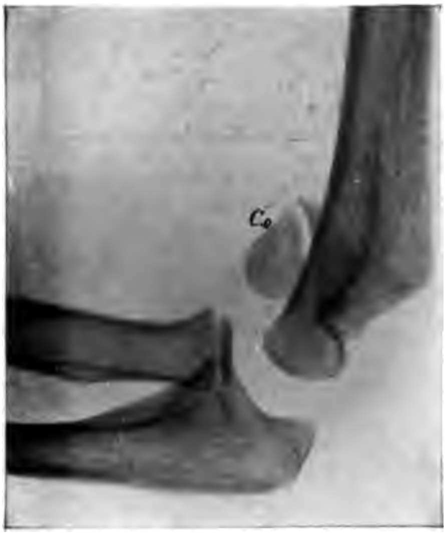


Fig. 348.—Fracture of external condyle. Seen from the side.

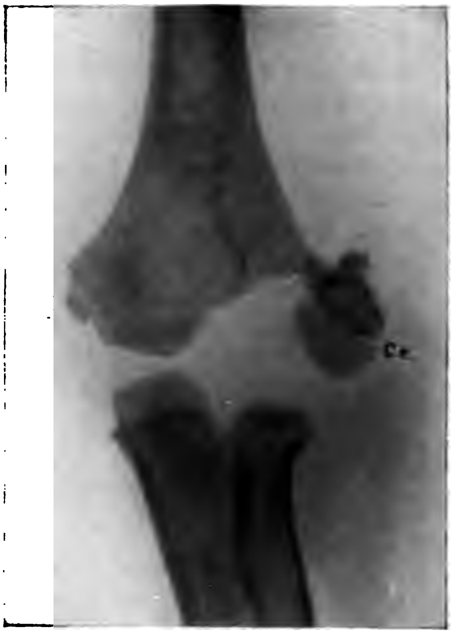


Fig. 349.—Fracture of external condyle. From behind.

(8) If the ordinary signs of a posterior dislocation exist, and if in addition crepitus and abnormal mobility of the external condyle or internal epicondyle are noted, it is obvious that the dislocation is associated with a fracture. This latter combination is a very typical occurrence.

(9) Sometimes, nothing may be recognized at first on passive movement and palpation; nevertheless, the impaired mobility and a

suspicion of crepitus being present, we cannot exclude a fracture. In some cases, pronation and supination are deranged, in some flexion is painful, and in others the derangements vary so much that they suggest a " dérangement interne "—although this is not a diagnosis. The following are the possible conditions.

(*a*) If there is localized pain on pressure over the **head of the radius**, if it is thickened and abnormally prominent, with a circumscribed effusion of blood in its vicinity, if pronation and supination are painful, or the latter movement quite impossible, the case is one of **fracture of the head itself** (chisel fracture, *Bruns* ; fig. 354).

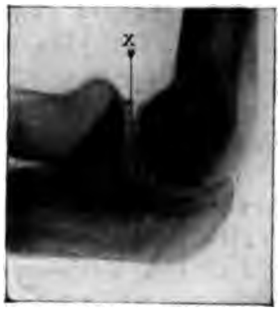


FIG. 350.—Detachment of cartilage and some bone (X) from the eminentia capitata humeri (see corresponding fragment in fig. 342.

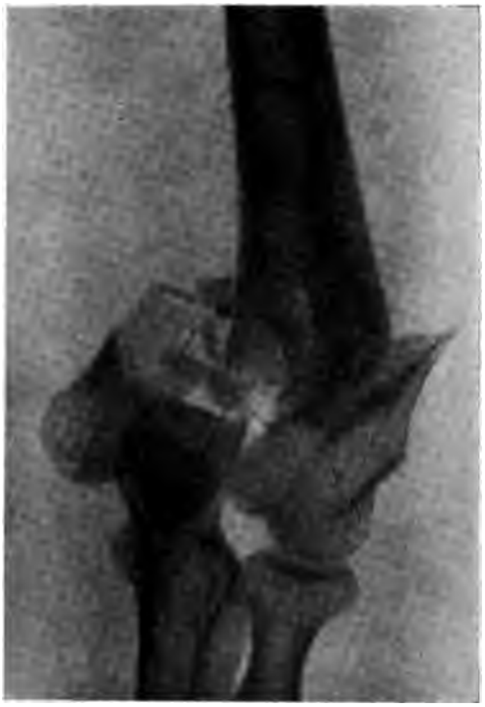


FIG. 351.—T-fracture of lower end of humerus.

Separation of the epiphysis is very rare, and it resembles fracture of the head of the radius, clinically, because the line of the epiphysis is in the head.

If the pain is in the neck of the bone rather than in the head, we must assume that the fracture is at the neck, either in the form of a wrench-fracture (Iselin, Stoecklein) a fissure or complete fracture of the neck. But even when completely fractured the head rests so firmly on the neck that it follows the latter on rotation. The detached head is sometimes turned to an angle of 90° so that its depression can be felt.

It is only possible to distinguish between fracture of the head and of the neck, if there is not much swelling—otherwise a skiagram is necessary.

(*b*) If the symptoms of a foreign body in a joint are the most prominent combined with some limitation of extension, we should think of an **abrasion fracture of the eminentia capitata humeri.** In this fracture, only a localized piece of cartilage with a small fragment of bone are broken off (fig. 350). This piece may sometimes be felt as a loose body, between the external condyle and the head of the radius, as soon as the arm is extended. On flexion, the piece of cartilage disappears within the joint. Sometimes nothing can be made out by palpation but the diagnosis can be established by a skiagram, as some bone is always broken off.

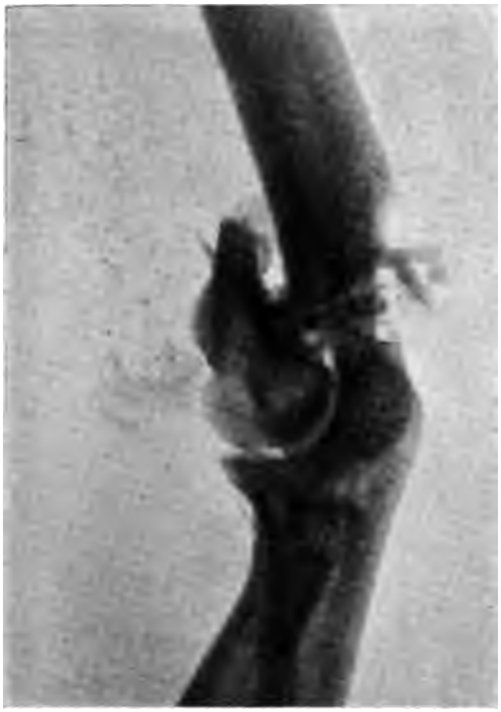


FIG. 352.—Supra-condylar fracture caused by a bullet.

(*c*) But if nothing is found, except that there is localized pain on pressure and ecchymosis of the elbow, we may assume that there is a **detachment of the coronoid process.** As soon as callus develops, it is very easy to detect this injury by palpation (fig. 356).

The diagnosis of "**sprain**" is only justified if an exhaustive and systematic examination fails to elicit anything definite.

IV.—RONTGEN RAY EXAMINATION.

We must never be content merely with a *screen* examination. The clearest picture on a screen fails to exhibit the details which a moderately good skiagram shows. It is impossible to avoid overlooking on a screen, subordinate, though important, injuries which may accompany the chief injury, e.g., a dislocation—an injury which is recognizable even without X-ray examination. Another precaution which must be observed, especially when dealing with growing individuals, is always to take a **control picture** *of the uninjured side*, in the same position as that in which we have examined the injured side. If this is neglected, we are liable to interpret the cartilaginous ends between the various centres of ossification as fractures of the

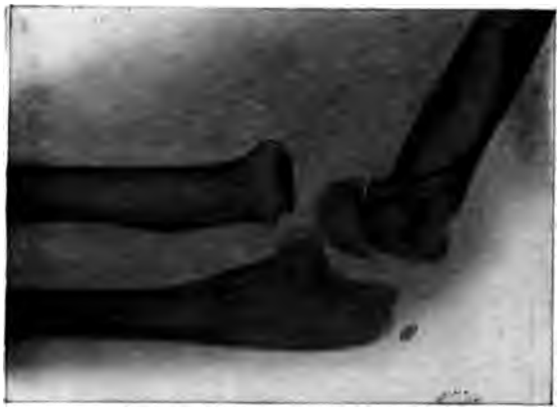


FIG. 353.—Forward dislocation of radius alone.

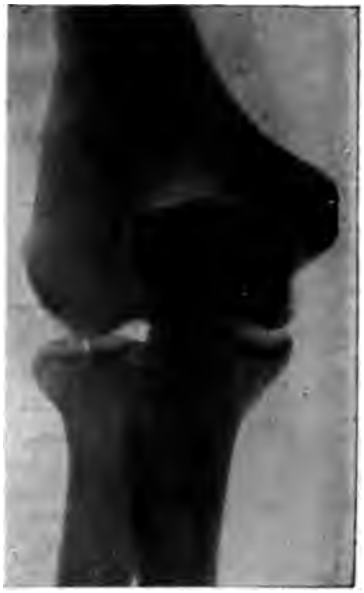


FIG. 354.—Chisel fracture of head of radius.

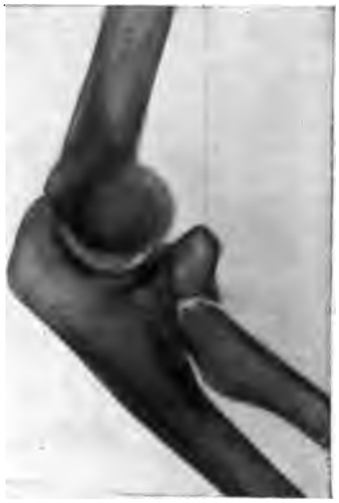


FIG. 355.—Detachment of head of radius.

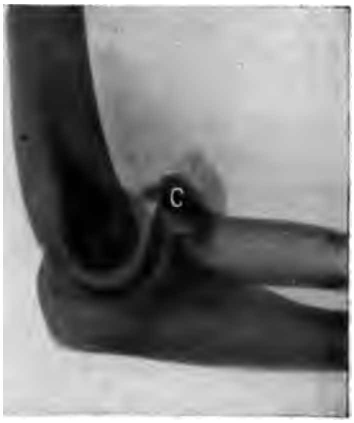


FIG. 356.—Detached fracture of the coronoid process of ulna. (C) Proliferation of callus visible above the fragment.

external condyle, the olecranon, &c., and in all probability we will overlook actually existing injuries. It is also indispensable to examine the joint in *two directions perpendicularly to one another*, from the front and from the side. The X-ray examination of an elbow injury is therefore not always an easy task, and the correct interpretation of the skiagram, especially in children, may be just as difficult as the accurate appreciation of what is found on palpation.

We must summarize the above in the following scheme—

No displacement of the cardinal landmarks (except sometimes in 7, when the olecranon is movable).		Moderate and transitory loss of power; nothing severe; pain on pressure: usually a little effusion.	(1) **Sprain.**
		Ditto, but severe pain on pressure over the head of the radius or its neck. The head often appears to be somewhat thickened; localized hæmatoma in its neighbourhood.	(2) **Fracture of the capitulum of the radius** (or detatchment of the head).
		As in 1, but severe pain on pressure in the flexure of elbow; possibly also swelling and crepitus therein; loss of power of active flexion (brach. internus muscle).	(3) **Fracture of coronoid process of ulna.**
		Joint free, but pain on pressure transversely above it.	(4) **Supracondylar fracture** without displacement.
		Loss of power variable; symptoms of foreign body in joint; loose body to be felt between rotula and head of radius, on extension.	(5) **Abrasion of eminentia capitata.**
		Severe loss of power and effusion; forearm can be displaced somewhat forwards and backwards (in position of dislocation). Some lateral displacement also possible.	(6) **Fracture through the condyles** (very rare).
		Passive movements free; active movements restrained; olecranon movable, sometimes proximally displaced.	(7) **Fracture of olecranon.**
Olecranon displaced in relation to the axis of the humerus, but not movable in relation to the ulna. The epicondyles not movable in relation to one another.	Displacement backwards.	Epicondyles not displaced; passive flexion restrained.	(8) **Posterior dislocation.**
		Epicondyles share in displacement, and movable in relation to shaft of humerus; passive movements free, or more extensive than normal.	(9) **Supracondylar fracture in hyperextension.**
	Displacement forwards.	Epicondyles not displaced.	(10) **Dislocation forwards** (rare).
		Epicondyles share in displacement forwards; passive movements free.	(11) **Supracondylar fracture in flexion** (rare).

Epicondyles or condyles separately displaceable in relation to the shaft of the humerus.	Internal epicondyle movable; usually displaced distally and dorsally.	(12) **Fracture of internal epicondyle.**
	Internal condyle movable.	(13) **Fracture of internal condyle** (very rare).
	External epicondyle movable.	(14) **Fracture of external epicondyle** (very rare).
	External condyle movable, usually rotated about 90° to 180°.	(15) **Fracture of external condyle.**
	Both condyles separately movable, in relation to one another and to the shaft of the humerus.	(16) **Y and T Fractures.**

CHAPTER LXXXV.

INFLAMMATORY PROCESSES ABOUT THE ELBOW.

(1) ACUTE INFLAMMATORY PROCESSES.

ACUTE inflammation of the soft tissues about the elbow may resemble an acute arthritis, just as in other joints. One should first think of **phlegmon of the forearm,** originating from a lymphangitis, which is so frequently the result of infected wounds of the hand. The etiology—i.e., a peripheral injury—at once indicates the correct diagnosis. It is not often that the elbow-joint is involved after these injuries. Apart from other symptoms, their chronological order serves to differentiate a superficial phlegmon from an acute arthritis. In the latter, pain and difficulty in movement appear first, and the superficial changes follow; in the case of a phlegmon, swelling and redness of the skin appear first, and the difficulty of movement later on. If the inflammation is limited to the antero-internal side of the joint, or at least has arisen in this situation, we may conclude with great probability that the phlegmon or the abscess has originated in the *glands of the elbow.* If the phlegmon has started behind, we look to the *olecranon bursa* for its origin. This bursa, like the pre-patellar, has a great tendency to inflammation, and the slightest skin abrasion in its vicinity suffices to afford entrance to the cocci and to cause an extensive phlegmon of the whole of the back of the elbow region. The more acute the process, the further it encroaches beyond the immediate limits of the bursa, and extends to the front and to the upper arm.

In contrast to this superficial inflammation, which at any rate at first leaves one side of the joint free, in **acute arthritis** the entire circumference of the joint is painful on pressure. The swelling is most evident where the capsule is most superficial, namely, about the radius and at both sides of the triceps tendon. But the soft parts in front soon swell up, and the whole region of the joint finally becomes red and œdematous. Reference should be made to the remarks on the shoulder-joint for the causes of the inflammation.

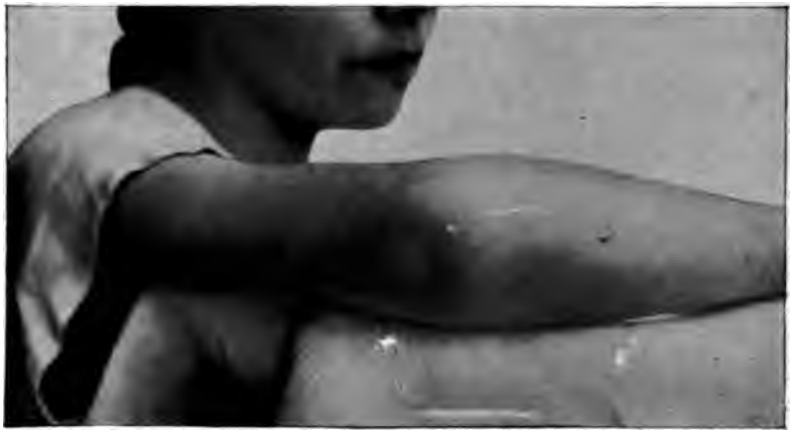


FIG. 357.—Tubercle of the elbow. Spindle-shaped swelling of the joint. Slight depression at the site of the triceps tendon.

(2) CHRONIC INFLAMMATORY PROCESSES.

Let us begin with the *soft tissues.* There are certain chronic inflammatory processes on the arm, the diagnosis of which is not clear at first sight. Examination shows that there is no primary disease of the underlying bone, and no portal of entry for organisms is evident. The changes consist of swelling and purulent infiltration of the subcutaneous tissue and of the skin, the brunt of the affection falling either on the one or the other. If any tubercular change, e.g., caries, disease of the tendon sheath, or lupus, be found on the hand, we diagnose the condition of the arm as one of *tubercular lymphangitis,* with its sequelæ, tubercular abscess of the soft tissues or tubercular destruction of the skin. Individuals suffering from this form of tubercle have very little resistance to the bacillus, and we therefore frequently find other foci of the disease present.

If there is no indication of this kind, we should think of the purely metastatic tubercle of the soft tissues, which is a rare form, or of *syphilitic gumma,* which is more likely, or of *actinomycosis,* which is

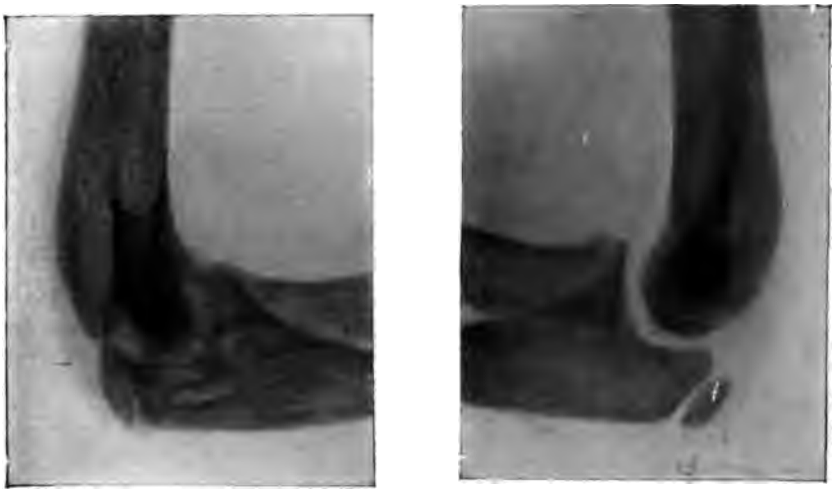


FIG. 358. FIG. 359.

Tuberculosis of the elbow.

(a) Diseased side. Cartilage has disappeared; bone eaten away, especially on the ulna.

(b) Healthy side.

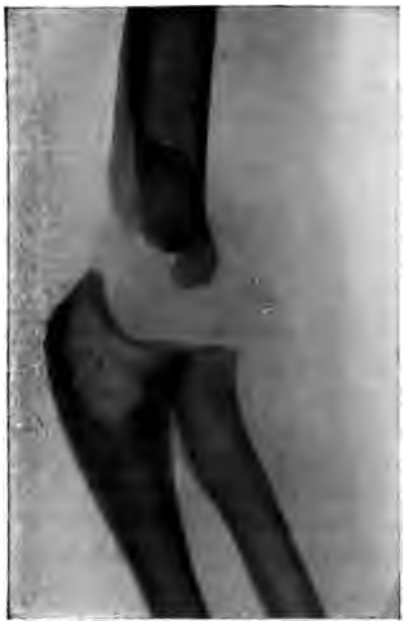


FIG. 360.—Tubercular focus in ulna.

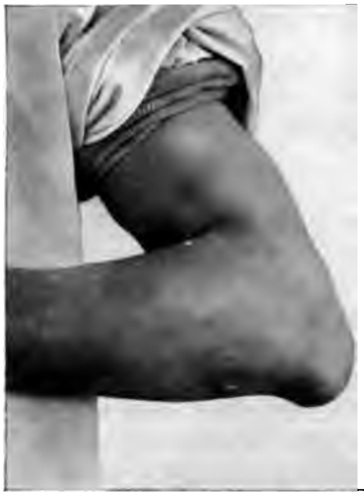


FIG. 361.—Chronic inflammation of olecranon bursa.

however, very rare, in the extremities. Until quite recently our diagnosis might have ended here. But within recent years it has been shown that a special variety of mycelial fungus (*Sporotrichum Beurmanni*) may cause persistent changes in the skin, the deeper tissues and even the bones, with signs intermediate between gumma and tubercle. This diagnosis of *sporotrichosis* has been made in many countries in isolated instances; but it can only be based on bacteriological examination of the pus—another reason for examining pus, the origin of which is not quite clear.

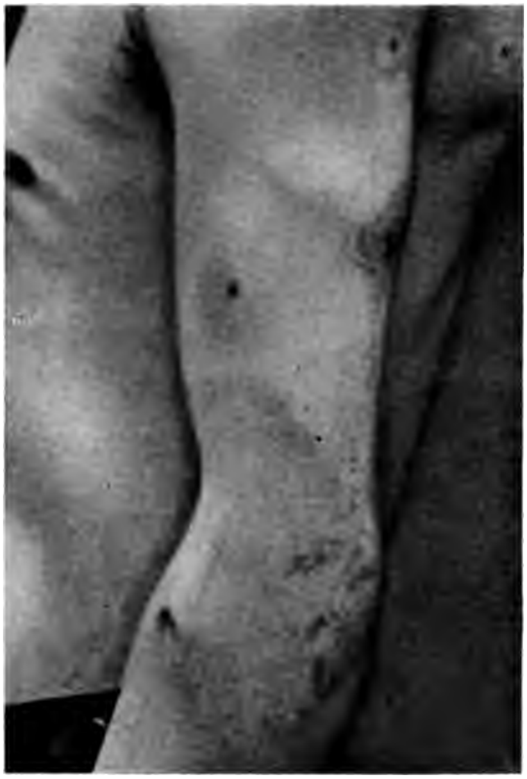


FIG. 362.—Sporotrichosis. Superficial pustules and deep abscesses.

Chronic inflammation of the elbow depends upon the same causes which we have encountered in chronic disease of the shoulder-joint. If several other joints are involved, we find in the term **chronic articular rheumatism,** discussed in that connection, a convenient diagnosis rather than a clear conception. If the elbow alone is diseased, it can hardly be anything but **tubercle.**

Careful palpation, comparing both sides, will generally detect the capsule, even if only slightly distended or swollen, in the form of two symmetrical pads (fig. 357) on both sides of the triceps tendon, and as a transverse pad at the level of the head of the radius. If the capsule is definitely thickened, the case must be regarded as one of tubercle, even if there be not much interference with movements. The axilla must always be examined for enlarged glands, although they may be less frequent in tubercular arthritis than in tubercular disease of the skin.

Whereas tubercle usually appears in the shoulder-joint in the form of a dry caries, without swelling of the capsule and without effusion, the fungating and caseous-suppurative forms occur most frequently in the elbow, both being accompanied by a moderate amount of effusion. The joint soon assumes a spindle-form shape, and the tendon of the triceps stretches at a slight depth between the two pads of capsule at the back of the joint.

It is but rarely that the skiagram indicates any osteoporosis (the purely synovial form). Definite foci in the bone (fig. 360) or superficial portions of the joint eroded away, are usually seen (fig. 358). In exceptional cases I have seen a certain amount of peri-articular formation of new bone, especially when fistulæ are present. If the bone formation is very extensive, it should raise the suspicion of syphilis.

A harmless chronic **inflammation of the olecranon bursa,** corresponding to the same condition of the pre-patellar bursa, must not be confused with a localized tuberculosis of the olecranon (fig. 361).

Occasionally one comes across persons who complain so persistently of pain in the region of the internal epicondyle, that one is inclined to think of tubercle. Sometimes a history of slight injury is given, but more often not. Nothing is to be detected, either by physical examination or by a skiagram. These cases have been termed *epicondylitis* (Francke) and the symptom has been attributed to traumatism of the insertion of the ligament and of the periosteum; and to slight inflammatory changes, e.g., after influenza, or rheumatism.

CHAPTER LXXXVI.

TUMOURS AND ALLIED SWELLINGS ON THE UPPER ARM AND FOREARM.

We encounter the same tumours and swellings in the upper extremity as we shall come across in discussing the lower limb, especially the thigh; but they are rarer in the arm.

There is nothing characteristic about the tumours of the *skin* and the *subcutaneous tissue* of the upper limb. Only lipoma of the shoulder (fig. 363) and the pendulous lipoma of the axilla (fig. 364) merit special mention.

Spindle-shaped tumours, following the course of a *nerve,* and originating in the *deeper soft tissues,* are usually **neuromata** or **neurofibromata,** but may be **sarcomata.** If the tumour becomes fixed on muscular contraction, we may assume that it has an *intramuscular* *origin*, in which connection we should think of an angioma of the muscle, of a sarcoma, of a gumma or of tubercle.

If the swelling can be emptied on pressure, or by elevating the arm, and fills up again when the arm is dependent, it is suggestive of an **angioma of the muscle.** These signs, however, only apply if the angioma is definitely of the cavernous type. But most of these r. curs contain a large amount of connective tissue and of fat, and smooth muscular fibres proliferate therein, so that they feel firm and even hard. The cavernous type is badly defined and extends in a diffuse manner; but the hard variety is definitely circumscribed and could easily be taken for sarcoma or early tubercle, if it were not for two contra-indicating circumstances—the long duration of the disease, and the attacks of the acute swelling (thrombosis) which the patients frequently describe.

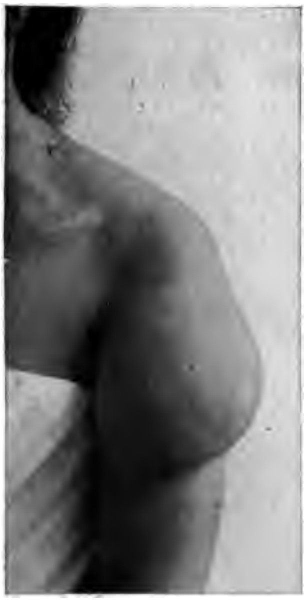


FIG. 363.—Lipoma of the upper arm.

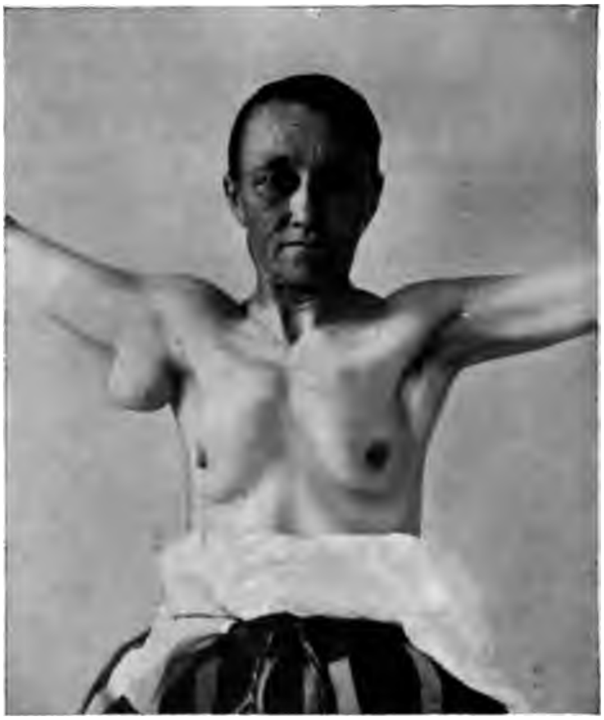


FIG. 364.—Pendulous lipoma of the axilla.

Tubercle of the muscle appears first as a small oval swelling which is definitely painful on pressure. As long as no suppurative softening and abscess formation bursting through the muscle occur, the diagnosis can only be one of probability, supported by a previous history of tubercle. If an abscess has formed, it is easier to recognize the nature of the disease; but nevertheless it may be impossible in some circumstances to exclude primary disease of the bone before the operation and without a skiagram.

A young man showed me a swelling, which had arisen a few weeks previously, on the anterior surface of the left forearm, in a position where there were no glands. Examination showed that the swelling was either intermuscular or intramuscular; family history revealed tubercle in a brother. Diagnosis: tubercle of muscle. The operation showed that the major portion of the palmaris longus had been converted into a tubercular area, which had not yet suppurated.

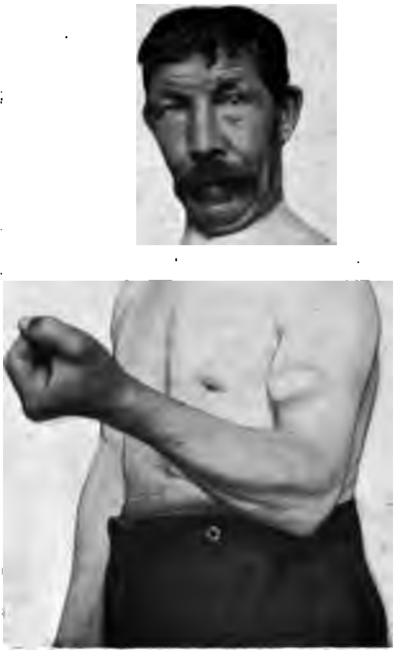


FIG. 365.—Hernia of biceps muscle.

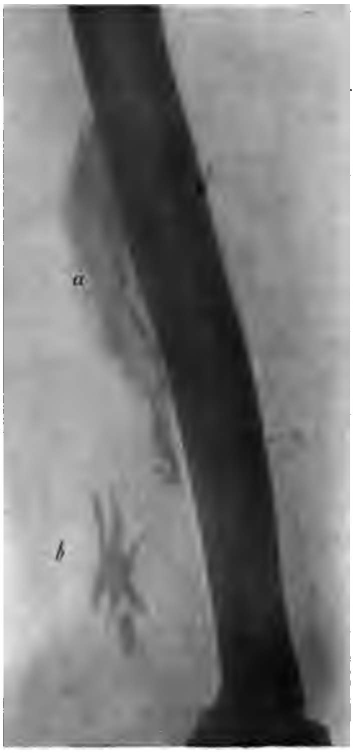


FIG. 366.—Intramuscular osteoma after a contusion. *a*, Connected with the periosteum. *b*, Lying free in the muscle.

If the tumour appears to be neither an angioma nor a tubercle, the question of **gumma** and of **sarcoma**, which are rare, or of **sporotrichosis** which is still rarer, will arise.

If we find a tumour over the biceps, resembling a pad transversely to the muscle, appearing when the muscle contracts and vanishing when it relaxes (fig. 365), it can only be a **hernia of muscle,** i.e., a firmly contracted mass of muscle projecting through a space in the aponeurosis.

This defect may be of traumatic origin; but it is occasionally bilateral and is probably then a congenital peculiarity. This was the case in the subject depicted in fig. 365, in whom the change was equally pronounced on both sides.

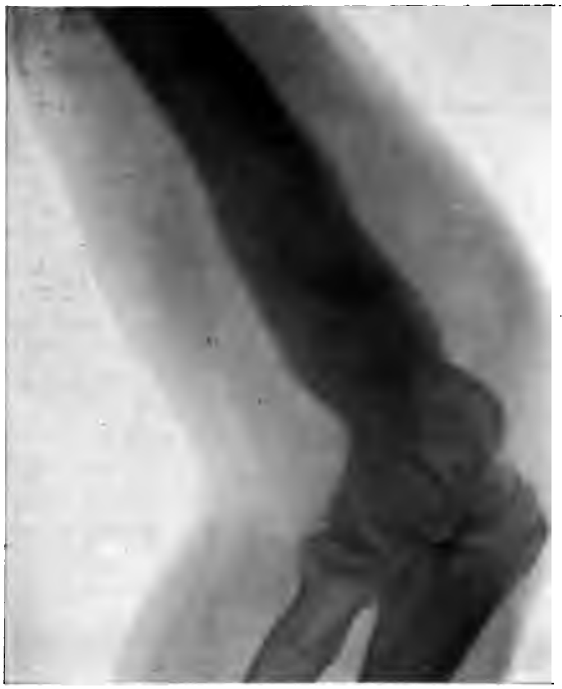


FIG. 367.—Gummatous periostitis and ostitis of the humerus and end of the ulna.

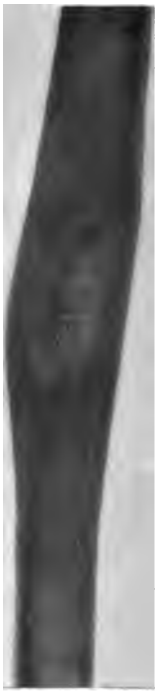


FIG. 368.—Chronic localized osteomyelitis of the humerus (abscess of bone and sequestrum).

If a tumour of bony hardness has developed after an injury—contusion of muscle, laceration of muscle after dislocation—we may assume the case to be one of **traumatic osteoma,** a circumscribed ossifying myositis. This is a process intermediate between new growth and inflammation, and occurs mostly in the brachialis internus muscle (fig. 366).

If a tumour is connected with the bone, and is increasing somewhat rapidly, it is necessary to differentiate between **tubercle, gumma,**

osteomyelitis and sarcoma; if the growth is slow, the diagnosis will lie between **sarcoma, fibroma, and chondroma.**

We should especially think of *tubercle of the diaphysis* in children. This condition can easily be mistaken for a chronic osteomyelitis, unless the pus is examined bacteriologically, both in its periosteal and myelogenous forms.

A *gumma* is diagnosed by the history of its comparatively painless course and by a skiagram.

I once saw a gummatous swelling which had existed for two years, and which I definitely looked upon as a sarcoma before ascertaining the history and having a skiagram taken. The patient, however, told me that he had had syphilis fifteen years before, and he himself attributed the swelling to this cause. He was right; the skiagram confirmed his diagnosis (fig. 367), and the swelling vanished after specific treatment.

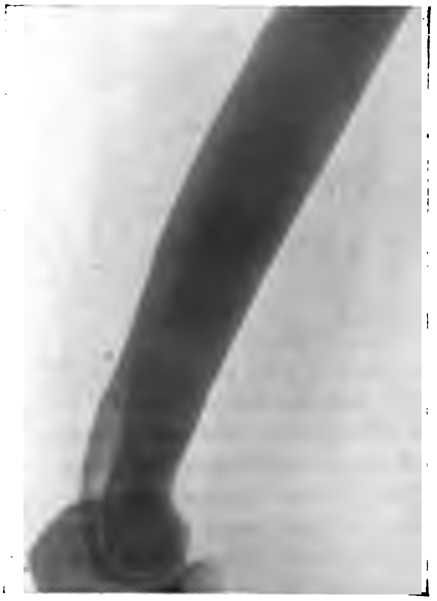


FIG. 369.—Old diffuse osteomyelitis of humerus.

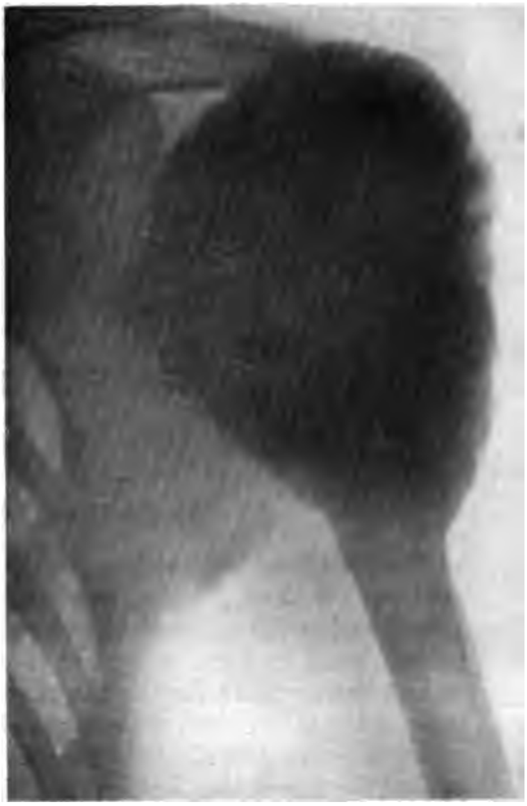


FIG. 370.—Osteo-sarcoma of humerus.

A *sarcoma* is usually first recognized when its circumference shows that it cannot be either tubercle or gumma. If the patient consults the doctor soon enough, it is quite possible that a skiagram would enable an early diagnosis to be made.

Distension of the bone, if very circumscribed, points to tubercle, but may also indicate sarcoma. Periosteal deposits in children occur in tubercle and in comparatively recent osteomyelitis; in adults almost exclusively in the latter condition and in gummata. Diffuse, smooth induration (fig. 369) or slight spindle-shaped distension points to old osteomyelitis which has run its course. The distension may still be concealing a sequestrum (fig. 368). Irregular proliferation of the periosteum must be ascribed to a gumma (fig. 367). A nebulous transparency of the bone in its entire thickness with the loss of details of its structure suggests a sarcoma. If there is, at the same time, considerable development of new bone, the diagnosis is osteo-sarcoma (fig. 370).

CHAPTER LXXXVII.

INJURIES OF THE WRIST AND HAND.

(1) RADIUS AND ULNA.

THE diagnosis of injuries of the wrist was a very simple matter before the time of X-rays. Anything which was not a fracture of the radius was a sprain, and vice versa. Dislocation of the wrist was looked upon as a curiosity, and it was asserted that the injury was so rare that it could not be diagnosed. The X-rays have shed some light upon this comfortable simplicity, but at the same time have raised new problems of diagnosis. In addition to fracture of the radius there are also numerous injuries and displacements of the carpal bones and their combinations. If these could only be recognized by X-rays, the matter would be no more difficult in practice than heretofore. Cases previously diagnosed as sprains would now be sent straightway to the radiographer as "injuries to the wrist," and nobody would take the trouble to attempt to diagnose a fracture of the radius. But fortunately these injuries can be diagnosed without X-rays, if they are properly examined. A skiagram should always be taken before ascribing the pain to exaggeration or malingering.

Fracture of the radius with pronounced fork-like posterior displacement need not detain us. It cannot be mistaken for anything else, if the classical symptoms are present, viz., displacement of the anterior fragment with the hand posteriorly and towards the radial side, freedom of the wrist-joint (fig. 372) and deviation of the styloid process of the radius with the wrist away from the axis of the radius (fig. 373).

The styloid process of the ulna is usually broken off in distal fractures (fig. 380, &c.), and the entire lower end of the ulna in more proximal fractures (fig. 378). This latter variety usually occurs in children and in old people with weak bones. Further back, there occurs the greenstick fracture which is so common among children (fig. 371).

If the fracture **involves the joint**, and the displacement is not very pronounced, the diagnosis is more difficult, for we have to take into consideration both sprains and injuries to the carpus.

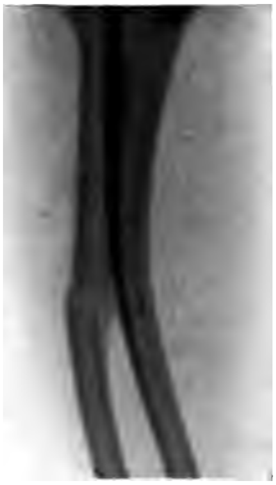


FIG. 371.—Sub-periosteal greenstick fracture.

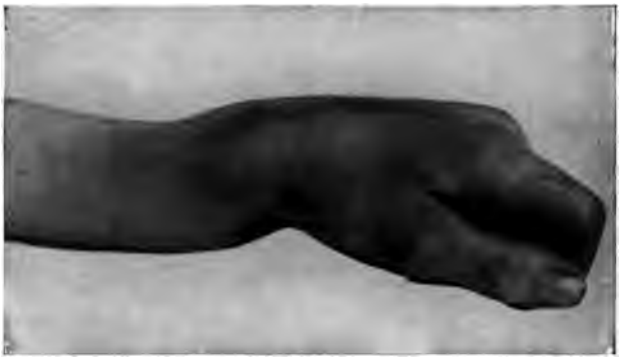


FIG. 372.—Fork-like posterior displacement, with fracture of radius (case of detachment of epiphysis with great displacement.)

If pressure in the long axis of the bone, care being taken not to move the joint, causes no pain, the injury is not likely to be a transverse fracture of the radius, if recent. But this pain on axial pressure does not persist after twenty-four to forty-eight hours, in cases of fracture without severe dislocation.

After this preliminary examination, we proceed to a more accurate palpation. If the styloid process of the radius or ulna is very painful on pressure, and if it is also somewhat thickened, we may conclude that there is a fracture, even if it is not possible to feel a movable fragment distinctly.

We then feel the radius carefully from before backwards, and as a control from behind forwards, ascertaining, point by point, the amount of pain on pressure. If there is no very pronounced pain, it is certain that there is no fracture. But if there is a position, on the elbow side of the end of the radius, wherein a definite and circumscribed pain on pressure exists, we must assume that there is a fracture of the radius, even if there be no visible displacement, which may, of course, be concealed by the general swelling. If this circumscribed pain on pressure can be traced over the whole width of the radius, the case is an ordinary transverse extra-articular fracture (figs. 374 to 379) ; but if the pain is only pronounced at the outer side and if, at the same time, there is some effusion into the joint, the case is one of an oblique fracture involving the joint (fig. 380). This variety of fracture, described by Barton some seventy years ago, was at that time the subject of considerable controversy. We now know, thanks to the X-rays, that this fracture is not at all rare.

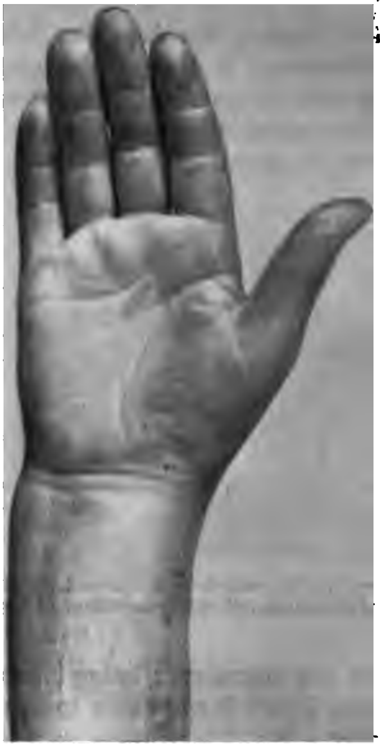


FIG. 373.—Deviation of hand from the radius in a case of fractured radius.

The presence of articular effusion, or at least of hampered activity of the joint, in association with more or less pain on pressure in a transverse direction, should raise the suspicion of a combined fracture, the ordinary variety of which is illustrated in figs 381*a*. These fractures also occur more frequently than was previously thought.

On the other hand, an isolated fracture of that portion of the end of the radius which is turned towards the ulna is very much rarer. Such a fracture may be suspected when there is a localized pain on pressure between the radius and ulna, and when the movement of rotation is painful.

A separation of the epiphysis must be thought of if the fracture is situated in the vicinity of the epiphyseal line in a young person.

We must refer to *another* variety of fracture, which occurs mostly among young people. This is illustrated in figs. 376 and 377. In these cases there is neither false mobility nor displacement. The loss of power is often so slight that no fracture is suspected. But careful examination will show that there is a sharply defined transverse area of pain on pressure, above the edge of the radius over the whole extent of the metaphysis. The skiagram reveals on either side a slight roof-like projection of the bone, showing that the radius has been compressed and that the crushed bone has been pushed out laterally because of the insufficient strength of the bone in its long axis. Iselin has shown that similar fractures occur in other metaphyses, but much more rarely.

If it is certain that a fracture of the radius is present, we must examine for the injuries which so often accompany this accident, namely, detachment of the styloid process of the ulna and fracture of the scaphoid (see below).

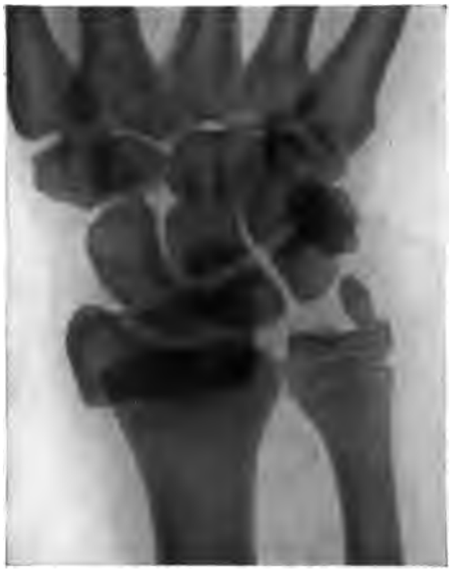


FIG. 374.—Separation of epiphysis of radius with displacement of radius backwards. Patient aged 19. (Case of fig. 372.)

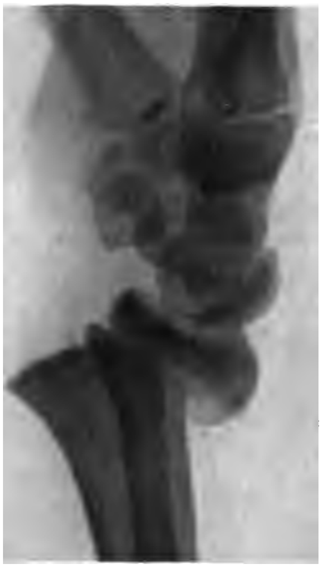


FIG. 375.—Same case, from the side.

(2) WRIST JOINT.

If nothing is found in the radius, we must consider whether the carpal bones have sustained any injury, or whether the case is one of a simple sprain. The typical examples of the former consist of palmar dislocation of the semilunar bone, fracture of the scaphoid, and a combination of the two.

(*a*) If we find, under the flexor tendons, a bony protuberance projecting towards the palm, or even only a marked thickening of the skeleton of the wrist in an antero-posterior plane, in a case wherein

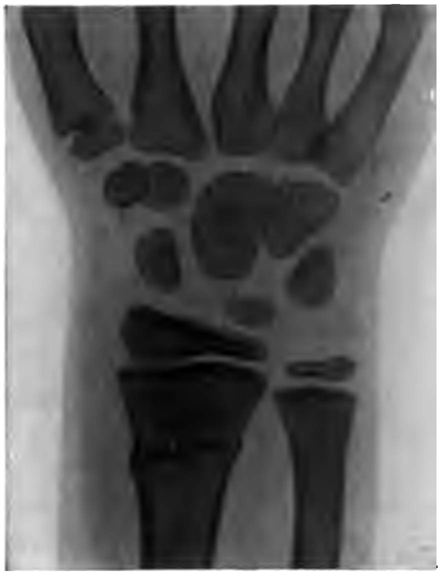


FIG. 376.—Fracture of radius, caused by axial pressure, in a boy aged 10.

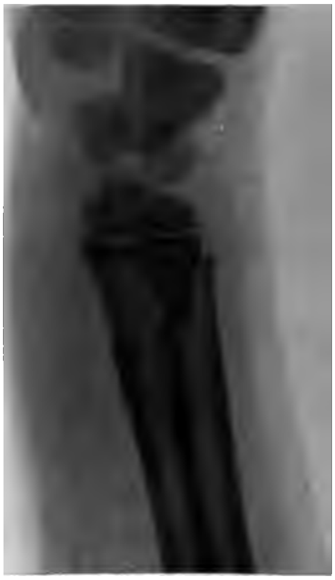


FIG. 377.—Same case, from the side.

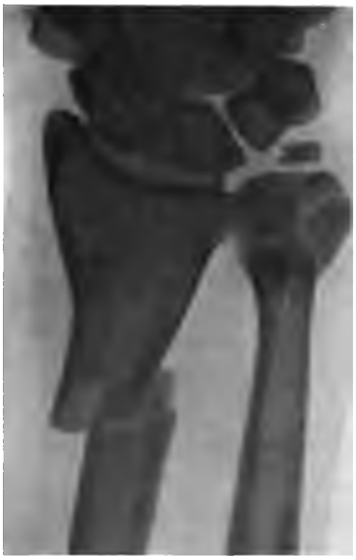


FIG. 378—Fracture of radius far back, with detachment and partial shattering of lower end of ulna.

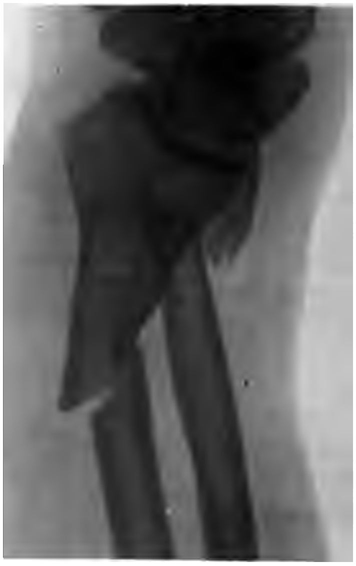


FIG. 379. Same case, from the side.

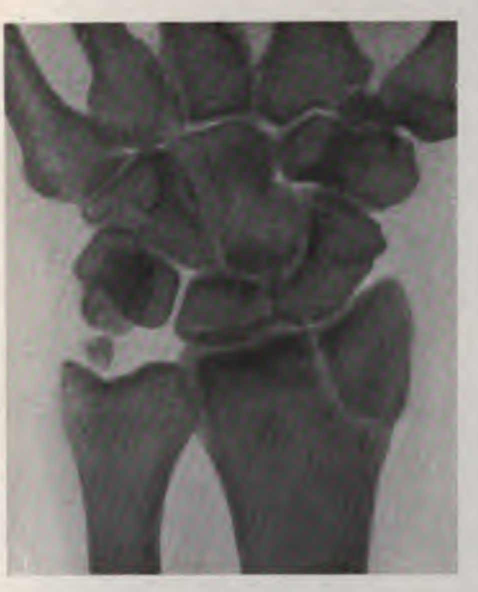


FIG. 380.—Oblique fracture of lower end of radius.

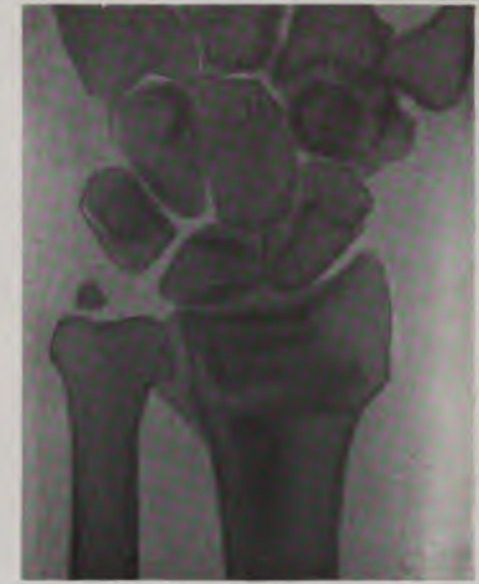


FIG. 381.—Typical transverse fracture of radius.

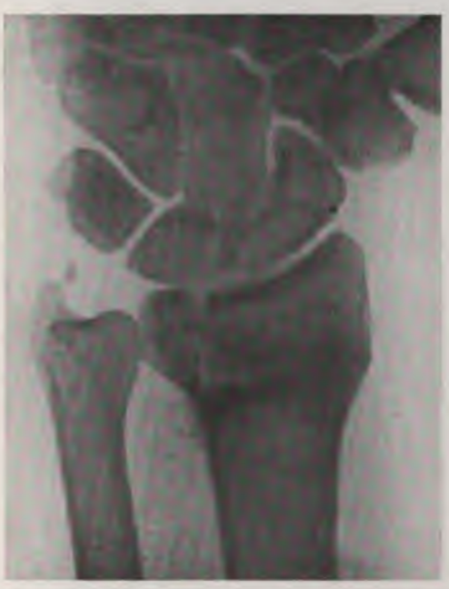


FIG. 381 a.—Combined fracture of radius, (Transverse fracture, with oblique fracture on the ulnar side.)

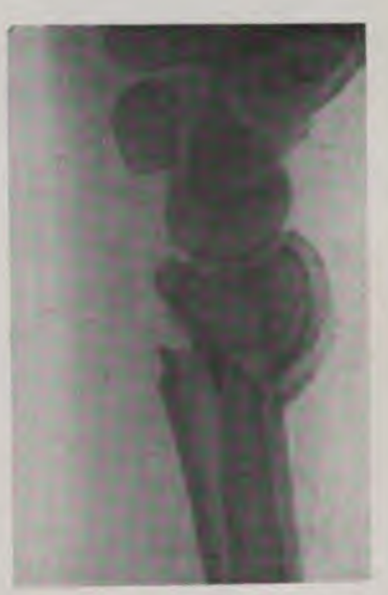


FIG. 382.—Dorsal deviation of the distal fragment in a case of fractured radius.

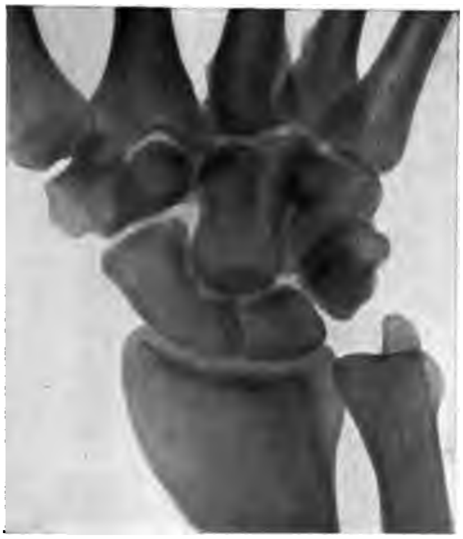


FIG. 383.—Normal wrist joint.

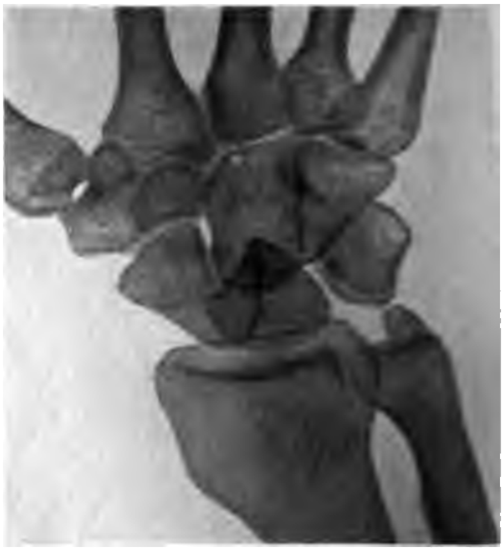


FIG. 384.—Palmar dislocation of the semilunar bone.

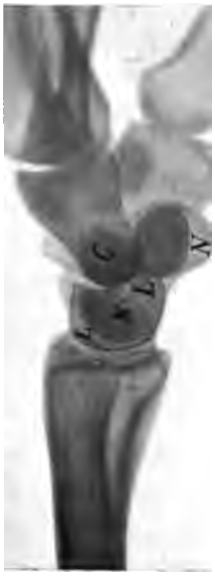


FIG. 385.—Normal wrist. *C* Os magnum. *N* Scaphoid. *L* Semilunar.

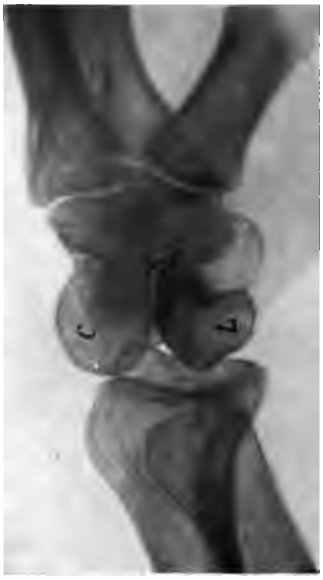


FIG. 386.—Palmar dislocation of the semilunar. References as in previous figure.

the joint is very painful and has lost its power, we must assume that there is probably a palmar **dislocation of the semilunar bone.** This diagnosis would be confirmed by the subsequent onset of neuralgia in the terminal fibres of the median nerve.

These injuries are nearly always recognizable in skiagrams taken in the dorso-palmar position. The semilunar is somewhat obliquely directed towards the scaphoid, and its distal articular surface looking towards the radius is easily detected (*cf.* figs. 383, 384). A skiagram taken in the lateral position always exhibits these injuries very clearly, and this measure should never be neglected in any obscure injury of the carpus. Such a skiagram shows the semilunar bone deviated towards the palm, with the head of the os magnum lying upon its dorsal surface (*cf.* figs. 385 and 386). In these cases the os magnum might be looked upon as dislocated just as well as the semilunar. Dorsal dislocation of the semilunar bone is very rare.

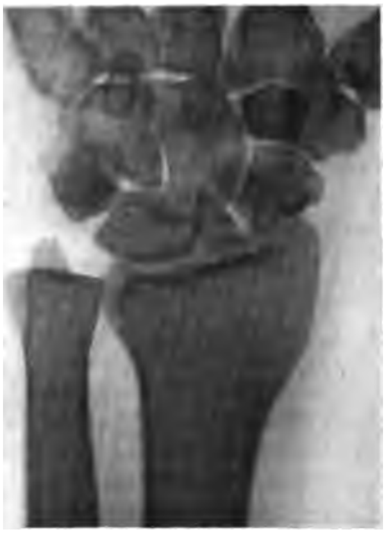


FIG. 387.—Transverse fracture of the scaphoid without displacement.

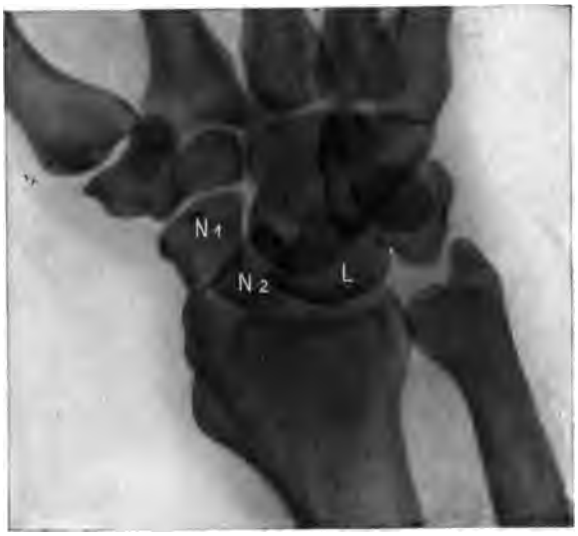


FIG. 388.—Typical intercarpal dislocation fracture. (Fracture of the scaphoid, with palmar dislocation of the semilunar L, and of the proximal fragment of the scaphoid, N$_2$.)

(*b*) If in addition to a moderate amount of palmar projection, there is definite pain on pressure over the scaphoid with shortening of the carpal region, and probably also some radial displacement of the hand, we must assume that the wrist has sustained a combined injury. This combined injury usually consists, as I have shown, of fracture of the scaphoid with palmar dislocation of the semilunar and the proximal fragment of the scaphoid attached to it. We term this for convenience the **typical intercarpal dislocation fracture.**

Fig. 388 is taken from the case, in which both sides were affected, which first suggested to me the typical character of this injury. Sometimes there is a transverse fracture of the os magnum combined with the injury; the styloid process of the radius or of the ulna may also be broken off occasionally. A definite fracture of the radius may even be present.

If there be no unusual bulging we must differentiate between fracture of the scaphoid, damage to the semilunar and a simple sprain.

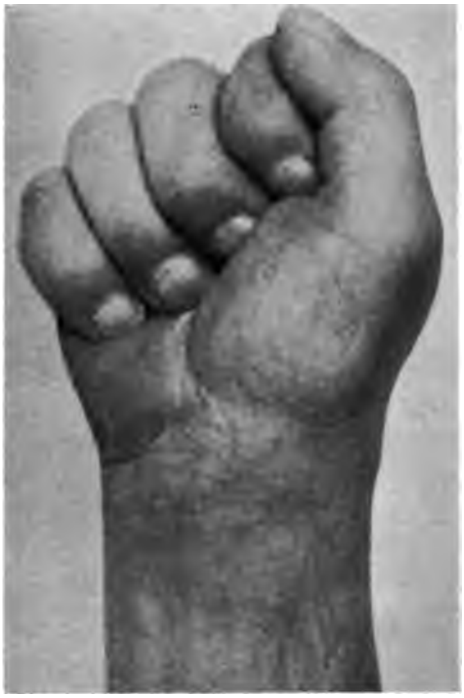


Fig. 389.—Typical intercarpal dislocation fracture, with visible projection on palmar surface.

Fracture of the Scaphoid—generally transverse without displacement of fragments—is indicated by definite pain on pressure in the tabatière. Absolute certainty can only be obtained from a skiagram.

We should suspect **damage to the semilunar bone**—usually a compression fracture—if the power of movement in the wrist joint is interfered with, and if there is pain on pressure over the semilunar; but here again a skiagram is required to be sure of the diagnosis (fig. 390).

The nature of the "damage" is still somewhat uncertain, but it appears that two varieties occur.

(1) Fracture or comminution of the bone by severe injury (a fall usually from a height).

(2) A gradual onset of atrophy of the bone with subsequent crumbling, probably due to nutritive changes following upon detachment of ligaments, usually the result of a slight sprain (Kieuböck, Guye). The clinical picture at first resembles that of a slight sprain, but the power of movement (especially dorsal flexion) does not recover and a skiagram taken a few weeks or months afterwards reveals atrophy of the entire bone with indications of bony tissue on the palmar and dorsal surfaces, or a central necrosis with a bony shell still retained at

the periphery. These bones, when removed by operation, have the appearance of osteitis deformans. It is quite easy to realize how these cases have hitherto been mistaken for tubercle—in the absence of any definite injury. These cases, whether treated or not, are liable to bring about degenerative changes in the vicinity of the semilunar bone, in the course of years.

We have hitherto confined ourselves to injuries which frequently occur in practice, and the diagnosis of which is important for the general practitioner. But there are, in addition, rare *dislocations* in the radiocarpal joint, in the intercarpal joints and carpo-metacarpal joints, some of which can be diagnosed by careful palpation, taking into consideration the position of the styloid processes, but which can all be definitely diagnosed by means of X-rays. It is unnecessary to discuss in detail any of these unusual injuries.

(3) THE METACARPUS AND FINGERS.

Fracture of the metacarpals can easily be distinguished from simple contusions by traction and pressure on the corresponding fingers. Only a longitudinal fracture would remain painless on such manipulation.

Fracture of the base of the first metacarpal may be mentioned as a typical, though not as a frequent injury. It is usually regarded as a sprained thumb, but the persistent pain shows that some more severe injury is present.

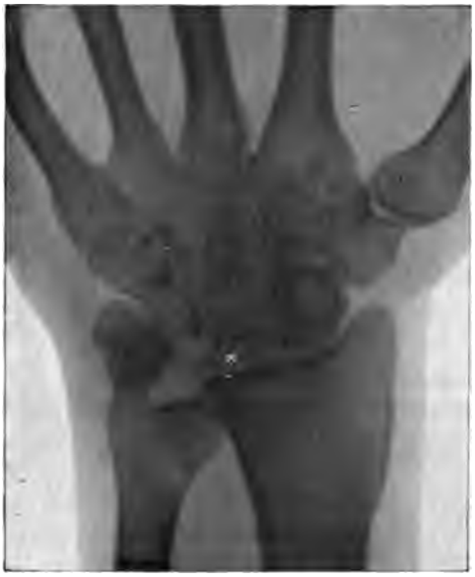


FIG. 390.—Contusion fracture of semilunar X (traumatic softening).

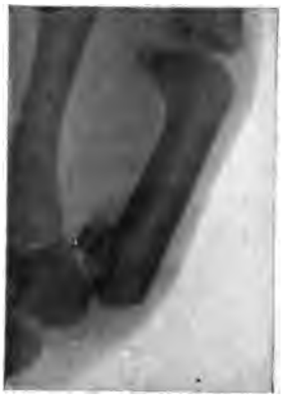


FIG. 391.—Fracture of the base of first metacarpal.

The skiagram shows either a transverse fracture of the upper end of the base (fig. 391), or a piece of bone broken off its palmar surface (Bennett's fracture).

The circulatory disturbances which follow injuries of the bones and soft parts are most pronounced on the *back of the hand*, just as in the case of inflammation. They are liable to persist in this position for a considerable time, even if they are not kept up by the manipulation of the patient who is insured against accidents. This condition has been termed "hard traumatic œdema of the back of the hand" (Secretan), but it has also been shown to be the result of percussion, practised by those who shirk work while seeking compensation.

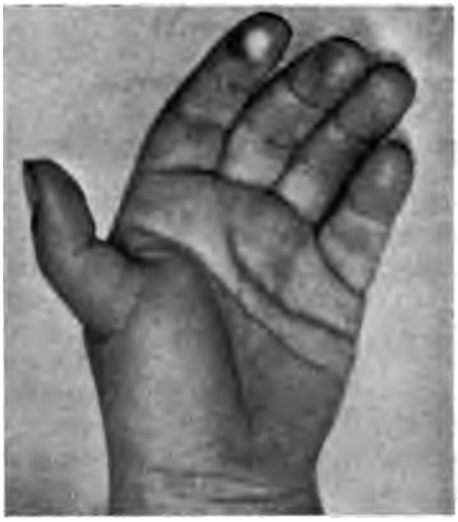


FIG. 392.—**Habitual dislocation of thumb.**

Fractures and dislocations of the fingers are so easy to recognize that they need not detain us. The same applies to dislocation of the thumb, so well known, because of the difficulty of its reduction. It is an injury which cannot be mistaken for anything else.

The following scheme summarizes the foregoing remarks on injuries of the wrist:—

No deformity of bone, evident on inspection or palpation.	**Radius nowhere showing any localized pain on pressure; carpus usually somewhat swollen; loss of power always pronounced; Röntgen rays always required.**	**Pain on pressure diffuse.**	(1) **Sprain of hand.**
		Localized pain on pressure in the tabatière.	(2) **Fracture of scaphoid.**
		Localized pain on pressure over the semilunar (middle of the back of carpus).	(3) **Contused fracture of the semilunar.**
	Radius painful on localized pressure, *behind* the line of the wrist-joint.	**Wrist free; the pain on pressure runs in a transverse direction.**	(4) **Extra-articular transverse fracture of the radius.**
		Wrist swollen; loss of power; pain on pressure close to dorsal edge of radius.	(5) **Fracture of radius extending into joint.**

Definite deformity of wrist.	Shape of wrist like the back of a fork, with the bend more towards the elbow.	.. ..	(6) **Extra-articular** fracture of radius, 3-4 c.m. or more behind the joint; usually with detachment of lower end of ulna.
	Shape of wrist like the back of a fork, with the bend near the carpus	Joint free.	(7) **Extra-articular** fracture of radius near joint; often with fracture of styloid process of ulna (also separation of epiphysis).
		Joint swollen, tender and stiff.	(8) **Intra-articular** fracture of radius (oblique fracture or transverse and oblique fracture).
	Dorso-palmar thickening of the joint without definite fork shape of wrist.	No definite shortening of hand; localized prominence of bone under the flexor tendons.	(9) **Dislocation of the semilunar** (or more correctly of os magnum).
		Shortening of hand; large mass of bone under the flexor tendons; pain on pressure over tabatière.	(10) **Inter-carpal dislocation-fracture** (fracture of scaphoid and dislocation of semilunar); not rare.

CHAPTER LXXXVIII.

INFLAMMATORY PROCESSES ABOUT THE WRIST.

(1) ACUTE INFLAMMATIONS.

We need only briefly refer to the severe inflammatory œdema of the back of the hand, which may follow any infected wound of the skin, either on the palm or on the dorsum. We must, however, deal in more detail with **inflammations of the tendon-sheaths,** wherein there may be some doubt at first as to the primary seat of the inflammation. If the patient states that the swelling has followed a perforated wound of the finger, or a bite, &c., we should at once think of the tendon sheaths, because such insignificant peripheral wounds frequently lead to acute suppuration within them.

The character of the loss of power furnishes an important differentiating sign. Inflammation of the tendon sheath especially interferes with the *movements of the fingers,* whereas disease of the joint affects the *movements of the wrist.* An inflamed wrist is painful all over, whereas in inflammation of the tendon sheaths the affected side only is painful. In acute arthritis, traction and pressure in the axis of the wrist is painful, but this is not the case when the tendon sheaths are inflamed. Finally, teno-synovitis always spreads in a longitudinal direction, whereas arthritis remains limited to the neighbourhood of the joint.

It sometimes happens that the joint becomes secondarily involved after a primary inflammation of the tendon sheaths. We may assume that such an event has occurred if pain, œdema and pyrexia persist, despite the opening of all superficial areas of pus, or if we feel and hear a sound of grating on moving the joint. This latter sign indicates that the articular cartilage has been partially separated or destroyed by the inflammation. On the other hand, the tendon sheaths may become secondarily involved as a result of disease in the joint or in the bones.

If the teno-synovitis has extended to the forearm, and is accompanied by fever, œdema and pain, we must carefully search for suppuration in order to make a timely incision into the abscess. As this is often situated deeply on the interosseous ligament, we must not wait for fluctuation before making a diagnosis.

Having diagnosed an **inflammation of the joint,** we must next determine its *nature* and its *cause.*

If other joints are also affected, and the inflammation subsides after the administration of salicylates, we may assume that the case is one of acute articular rheumatism. If only one joint is affected, and the salicylates are ineffective, gonorrhœa is the most probable cause, even if an injury is invoked as a pretext, or has actually happened.

An hotel servant sought to claim his accident compensation because of an acute swelling of his wrist, having sprained it in lifting a portmanteau. When asked whether he had had gonorrhœa, he at once gave the anticipated reply. The lifting of the portmanteau was merely the occasion of the first appearance of symptoms of inflammation; although, of course, the sprain may have favoured the attack of the gonococci on the joint. The patient should produce the most irrefutable evidence of an accident before an injury can be made responsible, even in a limited manner, for such an arthritis.

For the rest we may refer to what has already been said in connection with the shoulder-joint. In a few cases the diagnosis must be made by the course of the disease. Acute articular rheumatism runs a rapid course and does not usually impair the power of the joint; gonorrhœa takes a long time to recover, often lasting for months. In some cases it leaves the joint free, in others stiff. Staphylococcic and streptococcic infection lead, as a rule, to suppuration and to partial stiffening. The joint affection is temporary and does not go on to suppuration, in certain cases of sepsis (Streptococcus viridans, etc.) associated with endocarditis. These cases can only be diagnosed by a bacteriological examination of the blood.

(2) CHRONIC INFLAMMATORY PROCESSES.

No difficulty usually attends the diagnosis of inflammation of the wrist, which is chronic from the start. If several joints have been affected symmetrically, the case is one of **Chronic articular rheumatism,** the varieties and causes of which have already been discussed in connection with inflammations of the shoulder-joint (fig. 393).

If, on the other hand, only one wrist is affected, there is no alternative but to assume that the case is one of **tubercle** (fig. 395). It is most important to recognize this condition in its early stages. If the extent of the movements at the wrist gradually becomes restricted, if those which are executed are painful, if there is also slight pain on pressure, and some muscular atrophy supervenes in the forearm, we should think of tubercle, even though there be no visible swelling. If there is swelling, the question as to its differentiation from **tubercular teno-synovitis** may arise. The clinical pictures of the two diseases are, however, quite different. In tubercle of the joint the whole wrist is thickened in a spindle-shaped manner, in advanced cases the hand is in a position of slight palmar dislocation with complete extension of the fingers (fig. 395). The pain on pressure is equally pronounced on both sides of the joint, and there is also pain on traction or pressure in the axis of the wrist, as well as on any attempt at active or passive movements of the joint. In teno-synovitis, however, the swelling is only on one side, generally

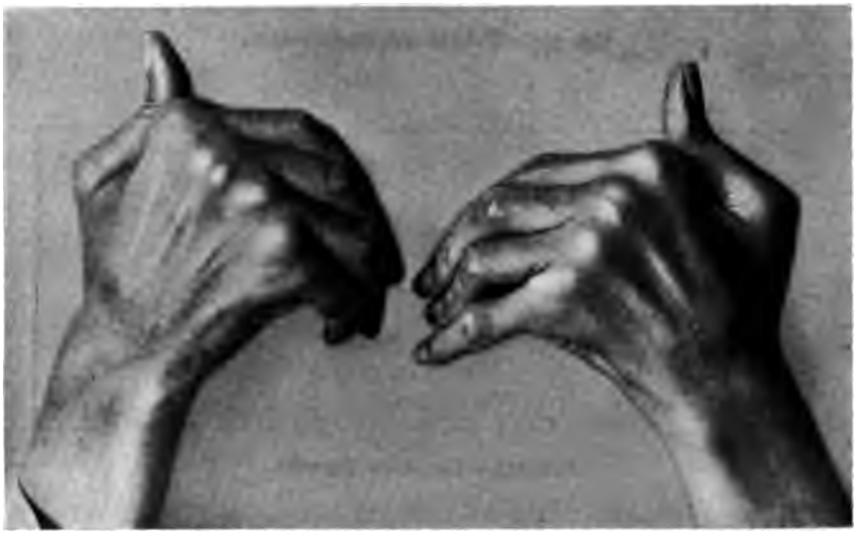


FIG. 393.—Typical posture of fingers in chronic arthritis deformans.

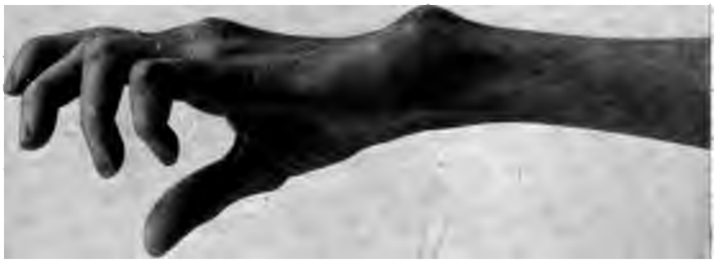


FIG. 394.—Wrist in arthritis deformans.

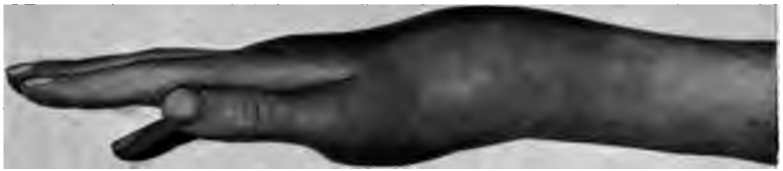


FIG. 395.—Tubercle of the wrist.

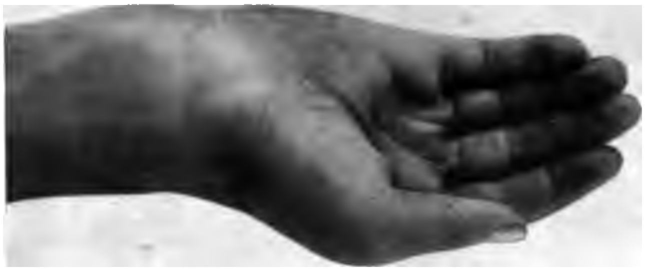


FIG. 396.—Tubercular teno-synovitis of the flexor tendons. Fingers slightly contracted in flexion.

on the palmar surface, and its maximum degree is not at the level of the wrist, where the tendon sheath is firmly bound down by the strong anterior carpal ligament, but either proximally or distally thereto (see fig. 396). The fingers are not extended, but are slightly contracted in flexion, and there is no palmar subluxation of the hand. If there be any pain on pressure, it is limited to the affected surface. The movements of the joint are only mechanically hindered by the swelling, but are scarcely painful. Pressure on the joint causes no pain.

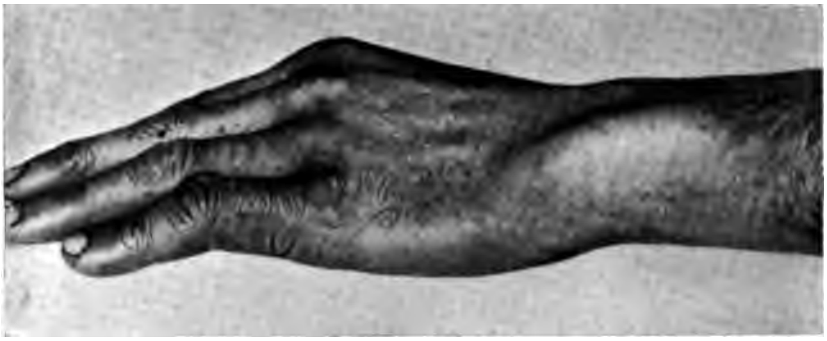


FIG. 397.—Chronic teno-synovitis of extensor carpi ulnaris.

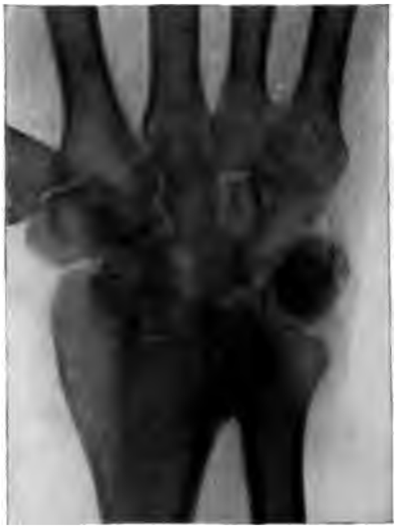


FIG. 398.—Arthritis deformans. (Skiagram of fig. 394.)

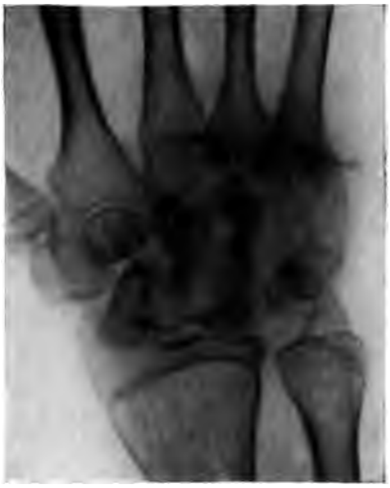


FIG. 399.—Tubercular arthritis.

If the joint is stiff and the movements of the fingers very restricted, and if fistulæ are present, it is neither necessary to inoculate guinea-pigs nor to take a skiagram in order to establish the diagnosis of tubercular arthritis.

A skiagram gives valuable information in all stages of the disease concerning the site and extent of the affection of the bone, and of the condition of the articular cartilage. In the early stage of synovial disease, X-ray examination only shows a diffuse osteoporosis, which differs by its greater uniformity from the acute maculated osteoporosis which occurs in acute inflammatory processes and in fractures (fig 400).

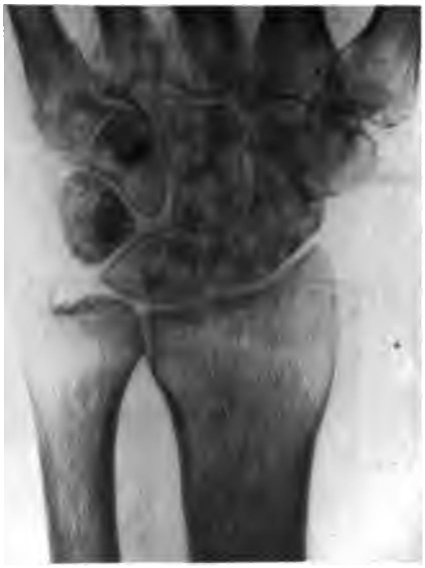


FIG. 400.—Acute maculated osteoporosis in a case of complicated fracture of the elbow.

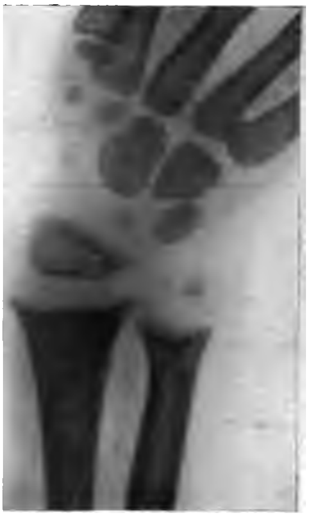


FIG. 401.—Rickety changes in bone.

If the disease starts in the bone, it can be recognized very early. In both forms the cartilage disappears in the course of the disease, and the individual bones come into direct contact. In the later stages the bones appear merely as shapeless and nebulous structures (fig. 399).

The extreme degree of bony changes which may exist in arthritis deformans is shown in fig. 398. It is interesting to compare with it a skiagram of a case of rickets (fig. 401).

CHAPTER LXXXIX.

ABNORMAL POSITIONS AND POSTURES OF THE HAND AND FINGERS.

1.—RESULTS OF INJURIES TO NERVES.

We have already referred on various occasions to paralytic conditions of the upper limb, which are of surgical importance. But it may not be superfluous to briefly summarize what has already been said, and to make a few amplifications.

Two questions arise in every case of paralysis :—

(1) Which muscles are paralysed ?

(2) Where is the interruption in the conducting path of the nerve ?

The reply to the first question only demands a knowledge of muscular function and a careful examination. Often a mere glance at the patient suffices for an approximate diagnosis, when the limb is held in a characteristic posture. If the hand hangs prone and lax, and the fingers cannot be extended, it is obvious that the case is one of *radial nerve paralysis.* If the thumb is extended against the index finger and cannot be moved away from this position, we should think of *paralysis of the median nerve,* and should test the power of abduction and flexion of the index and middle fingers especially. If this power is lost, and if at the same time the sensibility of the dorsal surface of the terminal phalanges is abolished, there can be no doubt about the diagnosis. If, on the other hand, the thumb cannot be actively approximated to the index finger, if the basal phalanges of the second and fifth fingers are slightly over-extended while the middle and terminal phalanges are slightly flexed, we should at once suspect *paralysis of the ulnar nerve.* It is easier to diagnose this condition at a first glance in the later stages, when the thumb, hypothenar eminence, and interossei are atrophied and the fingers have assumed the well-known posture of "main en griffe," i.e., over-extension of the basal phalanges with severe flexion of the middle and terminal phalanges (fig. 409).

If the patient cannot actively raise his arm at the shoulder, though the movement can be performed passively without difficulty, we should think of paralysis of the circumflex nerve, and should test whether there is any loss of sensation in the area to which the sensory branches of the nerve are distributed (over the deltoid muscle).

The second question, relating to the *site of the damage,* is, however, of greater importance from the point of view of surgical treatment.

This is frequently quite clear from the original cause of the disturbance (e.g., aneurism, tumour, &c.), or from the position of an injury, such as a cut, stab, or fracture of bone. Cuts over the wrist, which frequently involve the ulnar or even the median nerve, and fractures of the humerus, which may sacrifice the radial nerve, are mainly responsible. At the level of the shoulder-joint the dislocated head of the humerus may bruise the circumflex nerve, or, more rarely, one of the large cords of the brachial plexus. In the supraclavicular region the plexus may be directly injured, or indirectly, by means of a fragment of a broken clavicle.

In the absence of any such indication, or in cases wherein the injury involves simultaneously several sections of the limb, we should always give the preference to the causation which is able to attribute all the paralysis to one individual lesion. An example will make this clear.

A workman was hit on the head and shoulder by a large block of stone. When we saw him a few weeks subsequently, we were particularly struck by the posture of the left hand, which corresponded to that of radial paralysis. A fracture of the upper third of the humerus, which had healed somewhat at an angle, appeared at first sight to furnish the required explanation. But further examination showed that not only did the paralysis concern the extensors of the fingers and wrist and the supinators, but that it also involved the deltoid muscle, and that there was loss of sensation over the area supplied by the circumflex nerve. All this made it evident that the circumflex nerve was damaged when the fracture of the upper arm occurred. But this did not explain the paralysis and atrophy of the supraspinatus and infraspinatus muscles, which were no less striking than the other paralysis. There must, therefore, have been some injury higher up, and, as a matter of fact, there was a badly united fracture of the clavicle, the peripheral end of whose central fragment exactly corresponded with the position of Erb's point.

We were obviously not dealing with separate paralyses of the radial, circumflex and suprascapular nerves, but with a contusion of the nerve cord composed of the fifth and sixth roots, between the clavicle and the first rib—the so-called Erb's point. The accuracy of this assumption was proved by the fact that the muscles whose nerve supply corresponded exactly with the fifth and sixth roots were completely paralysed, i.e., the supraspinatus, infraspinatus, deltoid, coraco-brachialis, brachialis anticus, supinator longus and brevis. The nerve fibres to the long extensors of fingers, which are given off much lower down, were, however, evidently less directly involved, and the corresponding muscles therefore suffered less damage.

Just as damage to the upper roots of the brachial plexus, causing **Erb's paralysis,** produces a fairly uniform clinical picture, notwithstanding certain irregularities, a similar result follows from damage to the lower roots, causing **Klumpke's paralysis.** In this condition, the paralysis of the small muscles of the hand, and the disturbed sensation in the region of the median and ulnar nerve, are combined with oculo-pupillary derangements, such as miosis, narrowing of the palpebral fissure and retraction of the eyeball. The more protected position of the lower roots usually prevents their exposure to injury, and therefore Klumpke's paralysis is more frequently encountered as a result of tumours or inflammatory diseases of the bone (e.g., spinal caries).

The patient has not always paralysis when he consults the surgeon. The latter often has more occasion to see paralyses of the upper extremity arise as a result of treatment. These include *anæsthesia paralysis*, *Esmarch's paralysis* and *crutch palsy*.

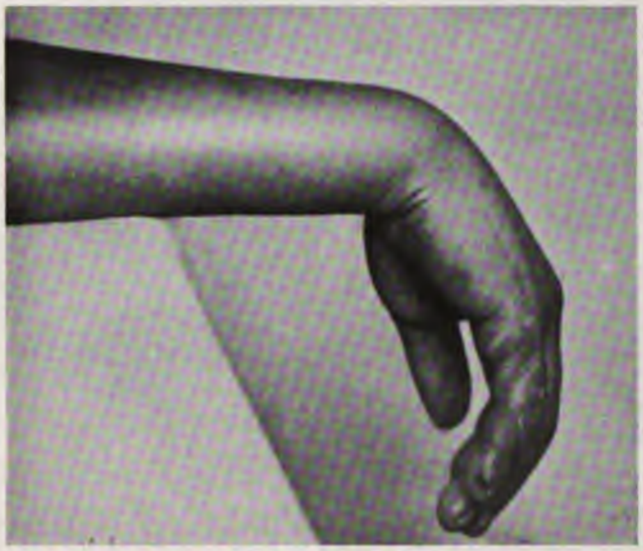


FIG. 402.—Paralysis of radial nerve, after a knife stab.

The first condition usually represents paralysis of the circumflex or radial nerve, and depends upon compression of the nerve-roots between the humerus and thorax, or between the humerus and the edge of the operating table, when the arm is raised. The mechanism of the two other forms of paralysis requires no further explanation. In all the three forms the motor fibres are usually alone affected, and spontaneous recovery occurs in a few weeks, or at latest in a few months. The course of ischæmic paralysis which comes on after too tight an application of a firm bandage, is quite different, because in such a case there is direct damage to the muscle in consequence of the deficient blood supply. The final result is not one of recovery but a fibrous degeneration of the muscle with contracture, in other words a permanent damage.

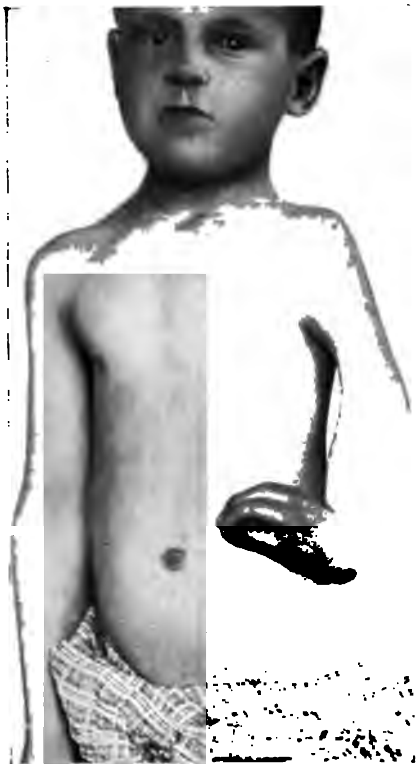


FIG. 403.—Manus vara. Absence of radius and of thumb.

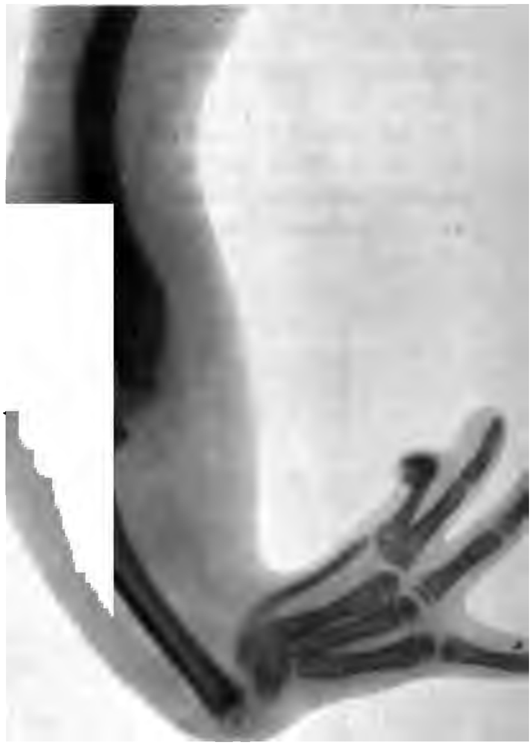


FIG. 404.—Skiagram of same case.

2.—ABNORMAL POSTURES OF THE WRIST-JOINT.

Abnormal postures of the hand are less frequent than those of the foot. Apart from ordinary traumatic deformities we distinguish congenital manus vara and acquired manus valga.

Manus vara, clubbed hand, which is generally seen in infants, always indicates a partial or complete defect in the radius. The thumb is often absent in these cases (figs. 403 and 404).

Manus valga, "Madelung's deformity of the hand," occur, not very infrequently, in females. The hand looks as if it had sustained a palmar subluxation. The lower end of the ulna is entirely displaced from its normal connections and projects markedly towards the

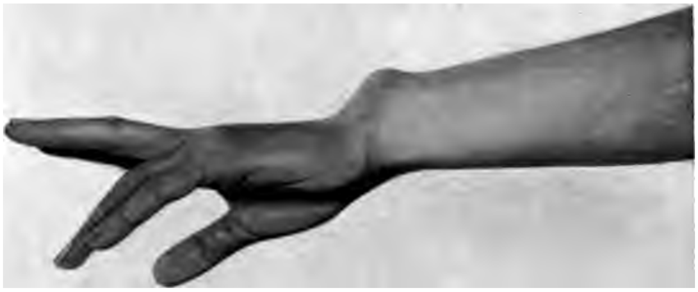


FIG. 405.—Manus valga. Madelung's deformity of the hand.

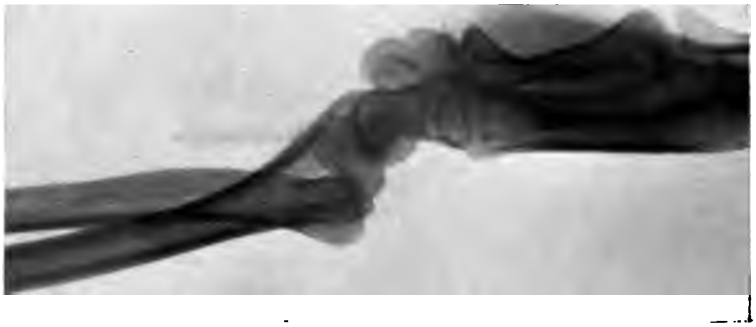


FIG. 406.—Radio-ulnar skiagram of the same case.

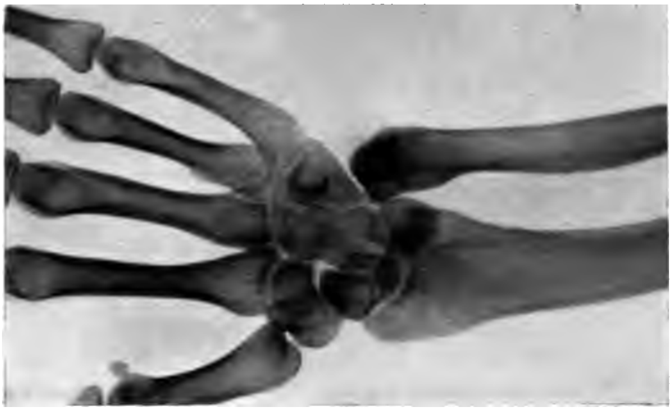


FIG. 407.—Dorso-palmar skiagram of the same case.

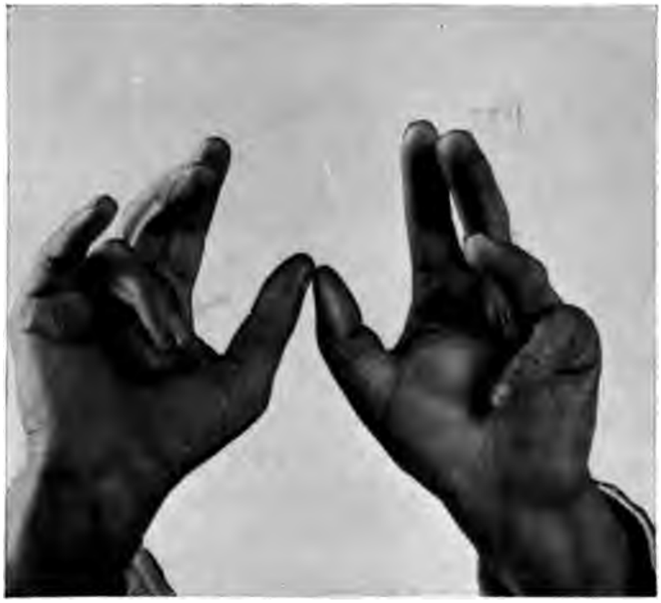


FIG. 408.—Bilateral Dupuytren's contraction.

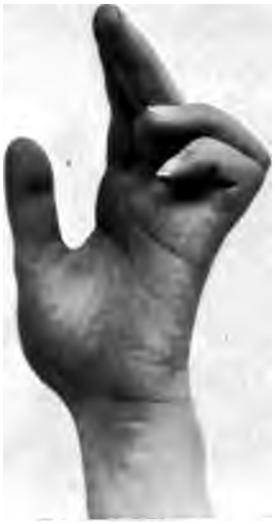


FIG. 409.—Contracture in ulnar paralysis.

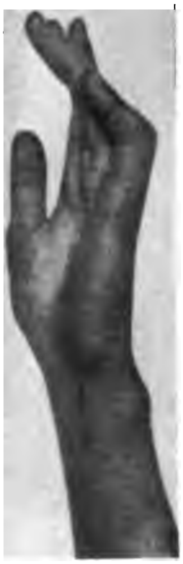


FIG. 410.—Congenital contraction of little finger.

dorsum. There is pain for a certain period of the disease, just as in genu valgum and pes valgus, and then it completely disappears.

The cause of this condition is not so much the result of occupation, as was originally thought, but is due to some change in the bone, depending upon curvature of the whole radius, resulting from late rickets. Its distal articular surface inclines towards the ulna and towards the palm, and thus allows the whole carpus to glide palmwards, causing a subluxation (figs. 405 and 406). The diagnosis is made at first sight.

It is but rarely that separation of the epiphysis due to injury gives rise to this kind of deformity. A traumatic origin might be suspected if the manus valga were distinctly unilateral.

3.—ANOMALIES IN THE POSTURE OF THE FINGERS.

We mention first among the anomalies of posture of surgical interest, the **bent little finger** which is sometimes hereditary, but is a cosmetic fault rather than a deformity. The anomalies of posture which may supervene after trauma and after tubercle of the bones are both innumerable and irregular.

Dupuytren's contraction of the **palmar aponeurosis** forms quite a characteristic picture. At first the patient merely notices that he can no longer fully extend his fourth and fifth fingers. On examination there will be found a localized remarkably hard thickening of the palmar aponeurosis running towards the affected fingers. The skin also exhibits swellings alternating with retracted areas. The disease usually involves both hands symmetrically, or one becomes affected soon after the other. Extension of the affected fingers becomes more and more difficult; the thick swelling continues to extend towards the hand and the fingers, the process involving one finger after another, sometimes even the thumb. The posture of the fingers is so striking that it cannot be overlooked or mistaken for anything else.

In rare cases an injury has been suggested as the cause; in other cases a tendency to gout and nervous influence have been blamed, and I have seen alcohol suspected in more than one instance. In the majority of cases the etiology is quite obscure. It occasionally happens that a contracture, which we must regard as Dupuytren's, is said to have come on after an injury in a case where there is some prospect of compensation. If the other hand also shows the beginning of a contracture we must not then attribute much significance to the unilateral injury.

We may also refer incidentally to the **trigger finger.** This symptom consists of the arrest of the movement of the finger in a certain position.

and its sudden advance with a jerk as a further effort is made. The symptom may depend upon some joint disease, e.g., on some abnormality in the shape of the articular ends due to injury or inflammation. But the cause is usually to be found in the tendon or tendon-sheath, and consists of a localized thickening, which produces mechanical obstruction in one definite position of the finger.

CHAPTER XC.

TUMOURS OF THE HAND AND FINGERS.

1.—INNOCENT TUMOURS.

MENTION should first be made of the **simple wart**, which is an infective but harmless papilloma of the skin. The illustration in fig. 411 is merely appended for the sake of contrast with the circumscribed form of warty tuberculosis of the skin (fig. 412). Innocent papillomatous changes in the skin sometimes involve the entire dorsum of the hand.

The most frequent tumour-like swelling is known as a **ganglion**. As we now know that these structures represent areas of gelatinous degeneration in the connective tissue of the joint capsule, and arise independently of the synovial sheath of the tendons and of the joint, we no longer trouble ourselves to differentiate between tendogenous and arthrogenous ganglions. They have nothing to do with tendon sheaths, but are closely related to the joint capsule, because they arise within its tissue. If there is any communication present, it must have arisen secondarily. It follows from the origin of the ganglion that its cavity is only separated from the joint cavity by a thin layer of connective tissue, which lies directly upon the synovial membrane. Unless, therefore, we proceed with the utmost care in extirpating a ganglion, we must be prepared for opening the joint over a limited area. There is, of course, no harm in this, if asepsis is maintained.

Some quacks tell their patients that a "nerve is displaced," in order to impress them with the belief that they are able to replace it. I know of a quack who provided every disease or injury of the limbs with this diagnosis, and, nevertheless, or perhaps because of it, had a large clientèle. Voltaire says, quite justly, that quackery started when the first swindler discovered the first fool.

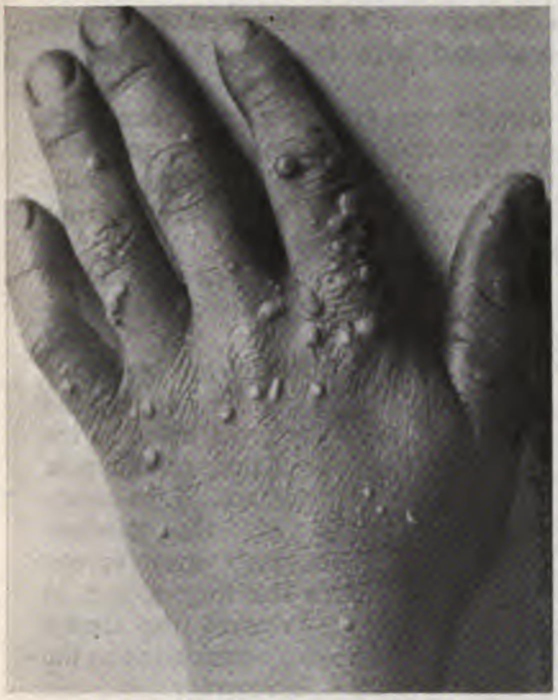


FIG. 411.—Simple multiple warts.

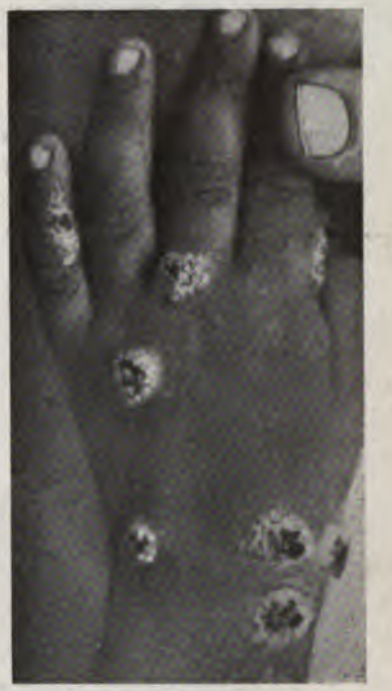


FIG. 412.—Tuberculosis verrucosa cutis.

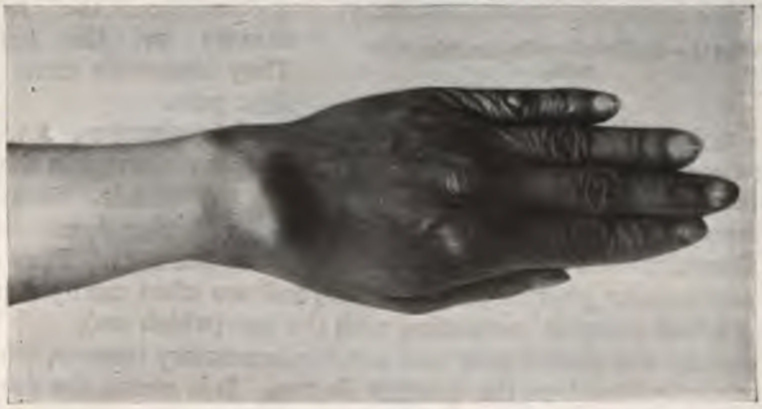


FIG. 413.—Ganglion of wrist.

One point is of diagnostic interest. It sometimes happens in cases of tubercular wrist, that *granulation masses of tubercle* protrude between the tendons as far as the skin, as visible separate tumours. Ollier has described cases wherein only a localized protrusion of the capsule has become affected with tubercle (tuberculomes juxtasynoviaux).

Cystic tumours of the hand and fingers are either **sebaceous cysts,** which are usually situated on the dorsum, or **traumatic epithelial cysts,** which are always found in the palm. The latter are generally considered to be due to some trauma which has displaced some epithelial cells into the deeper tissues (fig. 414).

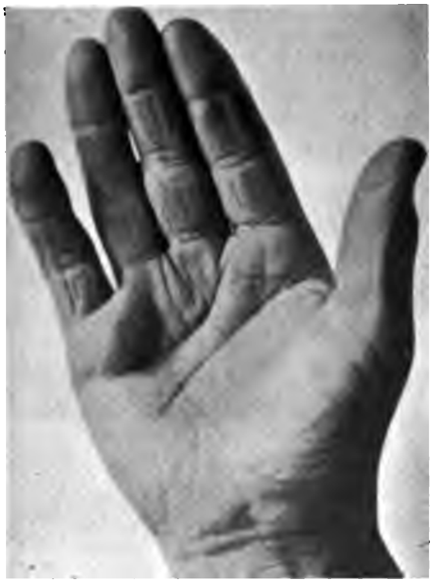


FIG. 414.—Epithelial cyst in the palm of the hand.

Franke thinks that some of these cysts are of congenital origin.

Lipomata are generally on the palmar surface, but they may grow between the metacarpal bones and appear on the dorsum. Like tuberculosis of the tendon-sheaths, they may spread towards the fingers, but in contrast thereto they always come to a termination as the carpal ligament.

Fibromata always present their usual characters : they are well encapsuled, are hard, and grow slowly in the tissue of the true skin, the palmar aponeuroses, the tendon-sheaths or the tendons. They frequently cause neuralgic pain.

Angiomata occur in every variety, as telangiectases, cavernous angiomata, and as circinate angiomata. They are situated in the skin, in the subcutaneous connective tissue or in the muscle. Sometimes an injury appears to be the cause of the origin of an angioma.

Chondromata have a very characteristic appearance. They occur as hard nodular growths of the fingers, and are often multiple, just as those which occur in connection with the toes (which see).

Finally, one should refer to a small inflammatory tumour, which is occasionally found on the hand or fingers. It is about the size of a pea, or somewhat larger, resembles a raspberry in appearance, has a thin stalk and is surrounded by a collar of epidermis. It is a granuloma, first described by Poncet and Bérard as **botriomycosis**, and is essentially a disease of horses. The one observer attributed it to a definite variety of *Staphylococcus botriomyces;* the other, to the *Staphylococcus aureus.* The appearance of the tumour is so characteristic that it cannot be mistaken if once seen. Histologically it is a telangiectatic granuloma.

2.—MALIGNANT TUMOURS.

The principal malignant tumours which occur on the hands and fingers are sarcomata and cancer of the skin.

Sarcomata have been seen on all parts of the hand, but most frequently on the fingers. They may arise in the skin, in the tendons or tendon-sheaths, or in the bones, in which latter case they may be mistaken for chrondromata.

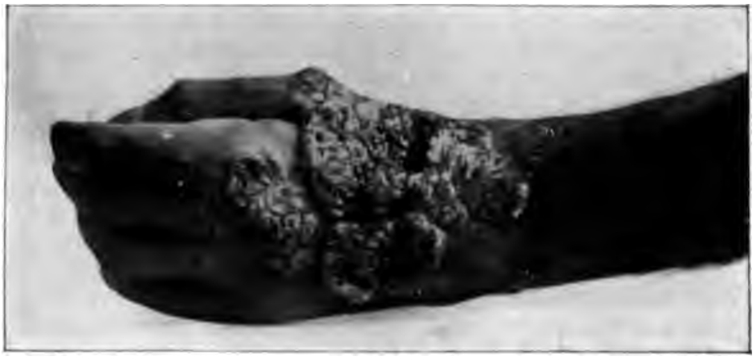


FIG. 415.—Cancer of the back of hand.

Cutaneous cancer always occurs on the back of the hand. It appears at first as a flat, more or less warty growth, which subsequently ulcerates extensively and assumes the usual characters of cancer (see fig. 415). Cutaneous cancer, arising from *chronic X-ray dermatitis,* or from *localized hyperkeratosis* independently thereof, deserves special mention, as many well-known radiographers have fallen victims to it.

CHAPTER XCI.

ACUTE INFLAMMATORY PROCESSES OF THE HAND AND FINGERS.

I.—INFLAMMATORY PROCESSES OF THE FINGERS.

ALTHOUGH inflammatory processes of the hand and fingers are routine matters of minor surgery, they do occasionally raise interesting problems.

If a patient comes with a swollen and inflamed finger, as a rule we diagnose a whitlow forthwith. But this does not complete our diagnostic task. Several diseases sail under the flag of whitlow, and we shall deal with them briefly.

We must anticipate, by insisting upon a careful examination for lymphangitis of the arm, and enlarged glands of the axilla, in every case of infective disease of the fingers. A trifling wound of the finger, which may have been healed within a few days, can lead to enlarged glands of the axilla and subsequent suppuration.

(*a*) **Dermatitis.**—A patient consults us for a severely swollen and inflamed middle finger, which looks more like a beetroot than anything else. He states that he had sustained a slight injury to the skin, which he treated by lysol fomentations by the direction of his doctor. The finger became swollen, and as the swelling increased, the more assiduous was he with the lysol fomentations. It is clear on examination that the two contiguous surfaces of the neighbouring fingers are inflamed, in addition to the middle finger. There is no extension of the inflammation to the hand or the arm in the form of lyphangitis; neither are there any general symptoms of infection. There is no sign of disease in the bone or tendon sheath, and the original wound is almost healed. Considering the entire condition, and especially the involvement of the two contiguous surfaces of the adjoining fingers, we are bound to assume that the case is one of **drug dermatitis.** The abandonment of all disinfectants and a dressing of simple ointment soon caused all the symptoms to disappear.

A similar condition may attend other disinfectants for instance corrosive sublimate, and especially iodoform. **Iodoform dermatitis** was a very common occurrence when the practitioner used to think that he had not discharged his duty adequately unless his patient reeked of iodoform.

Where the infective inflammation is deeply seated, the skin is tense and elastic, the epidermis smooth and shining. In drug dermatitis the superficial epidermis is infiltrated, uneven and rather rough, and is often raised by numerous little definite vesicles, or even by large blebs.

I once saw a slight wound of the finger, treated by sublimate compresses to prevent infection, which resulted in bulbous dermatitis reaching to the shoulder. The whole arm resembled an enormous sausage and was covered all over with blisters. In this case also the patient applied the compresses the more diligently as the dermatitis increased.

If the infection is deeply situated the patient complains of a stabbing, boring, aching pain, which prevents any rest, either by day or night; in dermatitis, however, the complaint is rather of a troublesome irritation and burning. In the former case local pressure causes great pain, in the latter case very little.

(*b*) **Primary inflammation of the bed of the nail.** If the inflammation starts superficially at a circumscribed spot and gradually spreads to the whole phalanx, the case is one of *infection of the nail-bed*, even if the bone necroses subsequently.

If the inflammation does not involve the bone, and nevertheless fails to subside in the ordinary manner, we should remember that a *primary chancre* has often been mistaken for a whitlow, and also that there is such a condition as *paronychia syphilitica*, in the secondary stage. If the patient exhibits a striking tendency to whitlows, although his occupation does not predispose towards them, we should examine for syringo-myelia (fig. 420), Scleroderma (fig. 421), Raynaud's disease, and diabetes. An ordinary whitlow, which runs a particularly severe course, is always suggestive of diabetes.

(*c*) An inflammation situated in the **subcutaneous cellular tissue** is distinguished from one due to primary disease of the bone by the fact that it is of very limited extent at first. If the accumulation of pus is not incised early, the inflammation may attack the tendon-sheaths, and then rapidly extend.

(*d*) We must also refer to **erysipelatoid inflammation** of the finger, which was described by Rosenbach, and more recently by Tavel, cases of which we have ourselves observed. Redness and hard swelling of the skin develop as a result of some insignificant wound of the skin, and the condition slowly spreads towards the hand, without leading to suppuration or causing general symptoms. In other cases lymphangitis, painful swelling of the axillary glands, and pyrexia occur. The disease is very liable to recurrence, and people who are occupied with meat, or animal offal, are the most frequent sufferers.

(*e*) We now come to **acute inflammations of the tendon sheaths.** These do not usually arise spontaneously, but follow some injury, extending as far as the tendon-sheath. Perforating wounds and bites are especially dangerous in this respect, because if they introduce septic organisms in deep situations, they are not easily dislodged, and have abundant opportunity of developing undisturbed.

If swelling of the finger occurs after such a history, the course of the tendon-sheath should be noted, and an immediate opening made, without waiting for definite fluctuation, if there is pain on pressure along the sheath.

Teno-synovitis is distinguished in its early stages from ostitis and periostitis by the fact that the inflammation is not limited to the course of one phalanx, and that the pain on pressure and the swelling are more pronounced on one side of the finger than on the other. The distinctness of the clinical picture often becomes obliterated in the more advanced stages, because a periostitis may develop from a teno-synovitis, and a secondary inflammation of the tendon-sheath may follow primary disease of the bone. If an injury can be excluded, *gonorrhœa* should be thought of. Gonorrhœal teno-synovitis usually begins very acutely, almost like a phlegmon, and then proceeds to a quiet chronic stage. Suppuration occurs especially in mixed infections.

If a manual labourer complains of a slightly painful swelling over the long extensor of the thumb, which has come on after hard work, and we feel distinct crepitation over the tendon and muscle, the diagnosis is *crepitant teno-synovitis*, a fibrinous inflammation of the tendon-sheath, the tissue around the tendon and the muscle.

(*f*) **Suppurative inflammation of the bone,** whether primary or secondary, can be recognized :—

(1) By the diffuse swelling and tenderness of the entire circumference of the finger, in the extent of one phalanx.

(2) By pain on axial pressure.

(3) By false mobility and crepitus in the adjacent joint, as the disease progresses.

A skiagram is of no assistance in the initial stage, but is very valuable later on, when the course is protracted and there is a development of new periosteal bone, or an involucrum begins to form, or if the case is somewhat more acute and the dead bone becomes divided from the healthy part by a light zone, even without the formation of any new bone (fig. 417).

(*g*) It is important to know something about **acute inflammation of the finger joints.** The first interphalangeal joint is most frequently affected. It acquires a spindle-shaped thickening, and looks somewhat like a radish in form. It is usually the consequence of some injury, and I have seen it particularly in butchers. The joint may remain extended with clear fluid for weeks after the subsidence of acute symptoms, even in mild cases. If the disease lasts for a long time, the cartilage finally disappears, as may be demonstrated by X-rays, before the joint yields any crepitus on movement. Gonorrhœa should be thought of if only *one* joint is affected, and if the disease has had a sudden and spontaneous onset.

The finger-joints may be involved *secondarily* in cases of suppurative teno-synovitis and ostitis. The clinical picture is, however, dominated in such circumstances by the primary disease, and the arthritis is merely a complication.

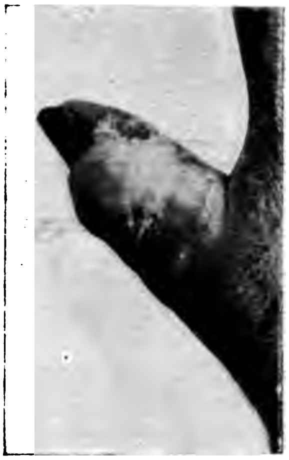


FIG. 416.—Whitlow causing secondary disease of bone. Infection of extensor tendon sheath by prick of a needle.

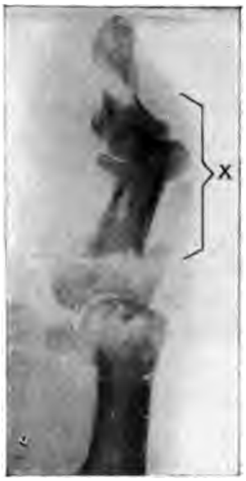


FIG. 417.—Skiagram of same. X = part of bone which has formed a sequestrum.

2.—ACUTE INFLAMMATORY PROCESSES OF THE HAND.

These arise from three different causes, leaving out of account the rare cases of **primary periostitis and osteomyelitis of the metacarpal bones.** They may originate as an extension **from the fingers,** as a result of **injuries to the hand,** or from suppuration of the **bursa,** which exists so frequently in the case of manual labourers, under the **callosities of the palm.** The diagnosis is usually very easy, but one must remember that even if the site of inflammation be in the *palm*, the œdema is most intense in the *dorsum*, because of the greater laxity of the skin. This peculiarity often leads the beginner to make his incision in the wrong place.

The practitioner often has to decide whether suppurative inflammation of the bursa beneath callosities is the result of an accident or not. If a wound of the skin, however minute, has led to infection, the decision is clear enough. But suppuration occasionally occurs without it being attributable to such a cause, and we are bound to regard it as a malady arising from occupation, and not as the consequence of an accident.

It should be mentioned that an **acute attack of gout** may exceptionally occur in the hand. An error in the diagnosis may lead to the hand being incised.

CHAPTER XCII.

CHRONIC INFLAMMATION OF THE HAND AND FINGERS.

THE skin, the tendon sheaths, the bones or the joints may be the seat of chronic inflammation in the hand or fingers.

(1) THE SKIN.

Chronic inflammatory conditions of the skin and subcutaneous tissue include primary chancre, lupus, leprosy, syringo-myelia, the trophic disturbances associated with Raynaud's disease and scleroderma, in addition to chronic eczema and to gumma, which latter is, however, of rare occurrence.

A **chancre** may be diagnosed by the history, early enlargement of the glands, and possibly by the secondary symptoms. Many a practioner has fallen a victim to a primary sore of the hand in the course of his profession.

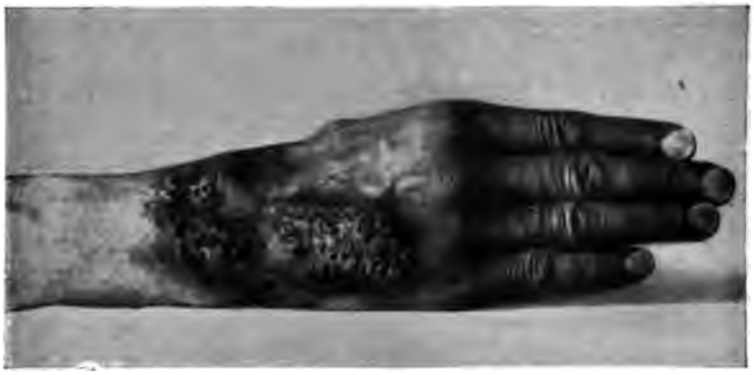


FIG. 418.—Lupus of back of hand. (Tuberculosis verrucosa cutis). Late syphilitic lesions are always in the palm, in contrast to most similar conditions. The illustration in fig. 419 is quite characteristic.

Practitioners who cannot restrain themselves from touching every wound or ulcer with their fingers, may be reminded that, even if they do not fear the organisms of suppurations, they may still be in dread of the spirochætes. A well-known dermatologist says, not unjustly, "Whoever touches every ulcer with his fingers, shows that he does not know what it may be, or that he has already had syphilis." If the ulcer must be touched, an india-rubber finger-stall should be used, for the protection of oneself and other patients.

Lupus, in its various forms, is mostly situated on the back of the hand or fingers. It is recognized by its usual characteristics, and we would especially refer to what has already been said in connection with lupus of the face, for the points of distinction between it and tertiary syphilitic lesions. Lupus may, in rare cases, ulcerate very deeply and even destroy the tendons, eventually causing severe contractures. If the tubercular process attacks the bones and joints, there may ensue an amount of destruction which is suggestive of leprosy.

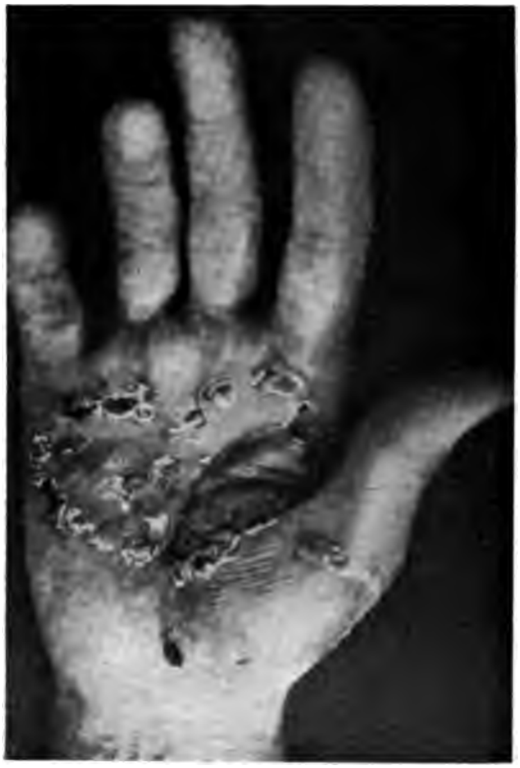


FIG. 419.—Papulo-ulcerative syphilide of the palm.

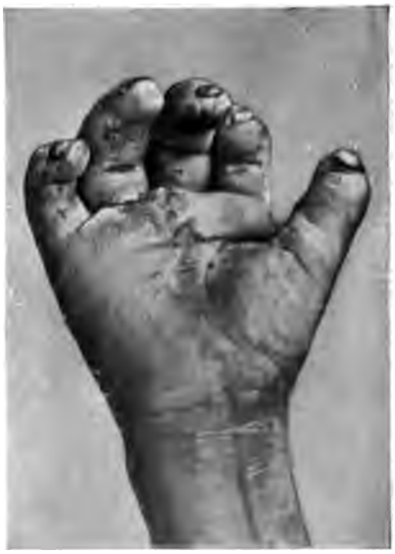


FIG. 420.—Mutilation of hand, due to syringo-myelia.

Post-mortem **tubercle** and skin tuberculosis of butchers should also be thought of, if the appropriate causes exist.

Leprosy of the fingers is chiefly recognized by the fact that it leads to their spontaneous amputation. If this condition exists we must at once ascertain whether the patient has lived in a leprosy district. In addition to the well-known regions of leprosy, there are numerous scattered centres of leprosy in rarely visited districts, which should be taken into consideration. In doubtful cases, we must search for traces of macular leprosy in persistent atrophic, superficial and cicatricial cutaneous changes in various parts of the body, and in thickening of the large nerves, especially the ulnar.

The mutilation in syringo-myelia and Raynaud's disease compete with that in leprosy. The symmetry and the associated nerve symptoms are always conclusive. The latter must, however, be carefully sought for, because the patient is often quite unaware of them (fig. 420).

(2) THE TENDON SHEATHS.

Inflammation of the tendon-sheaths, which is chronic from the start, and which is accompanied by swelling, is, practically without exception, of a tubercular nature. The flexor tendons are most frequently affected.

I once saw a case of extensive tubercular teno-synovitis of the extensor tendons in a butcher, who had wounded himself on the corresponding place fifteen years previously with a splinter of bone of a tubercular cow. An old scar still remained in evidence of the wound.

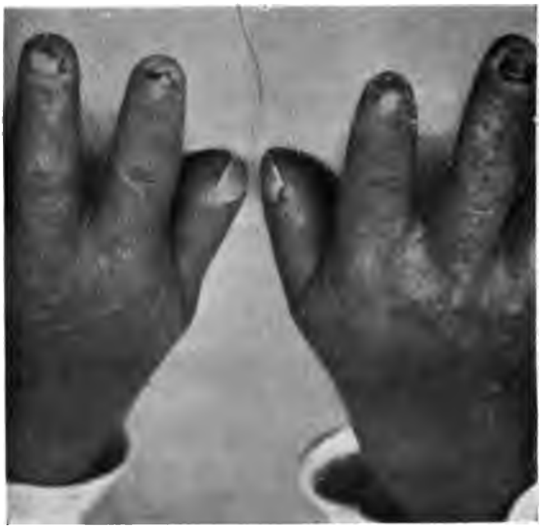


FIG. 421.—Scleroderma.

Tubercular teno-synovitis is easily recognized by the puffy induration in the region of the tendon-sheath and by the stiffness of the corresponding finger on slight flexion (fig. 396). The common tendon-sheath under the anterior annular ligament is occasionally affected, and the disease extends therefrom in four processes to the second, third, fourth and fifth fingers. There is frequently no fluctuation, or it may be more or less clearly recognizable in the palm only. If there is any considerable effusion the sac is subdivided, being constricted by this ligament just mentioned. The fluid can be displaced from the palm to the forearm and vice versa. Crepitation indicates the formation of melon-seed bodies. At first, the tendon sheaths only are affected, but spindle-shaped areas of granulation tissue with separation of the tendon-tissue into brush-like masses, may develop in course of time. Chronic enlargement of the axillary glands confirms the diagnosis, if confirmation should be necessary.

Tubercle could only be mistaken for the subacute stage of *gonorrhœal teno-synovitis* or for the much more rare *lipoma of the palm*. The former would be indicated by a sudden onset with severe pain ; the latter by a painless onset.

We must still refer to another malady, which may cause the patient much agony, although it is trifling, and easy to relieve. This consists of the relative narrowness of the compartment of the tendon-sheath, lying on the styloid process of the radius, which transmits the extensor pollicis brevis and the abductor pollicis longus—a condition which I first described in 1895, at Kocher's suggestion, as **contracting teno-synovitis.** It is not an inflammation in the strict sense of the term. The patients, who are mostly females, complain of pains radiating towards the thumb and forearm on any effort. On physical examination, the only thing to be noted is a striking tenderness on pressure, and sometimes a slight swelling in the vicinity of the above-mentioned compartment of the tendon-sheath.

If the tendon-sheath is exposed under local anæsthesia, the tendons are seen to be constricted within it. The sheath should be split, which may also be done subcutaneously with a tenotome, and the patient is immediately and permanently cured. Histological examination merely shows thickening of the wall of the sheath, without any inflammatory changes.

(3) THE BONES.

A spindle-shaped swelling of a metacarpal bone or a phalanx, which has developed gradually and with little pain, and which eventually suppurates and forms a sinus, is almost always **tubercle.** We say *almost*, because there is a very similar condition of the

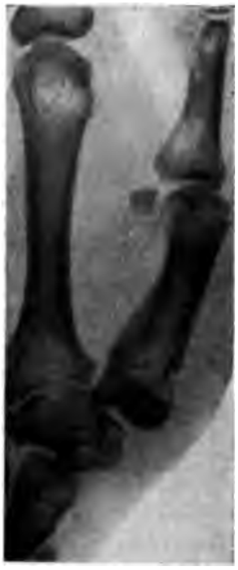


FIG. 422. — Syphilitic osteitis of 1st metacarpal.

phalanx, which is due to **syphilitic dactylitis.** But the mere diagnosis of tubercular disease does not exhaust all that is necessary to know. From the point of view of prognosis, it is important to ascertain whether the disease has started in the *medulla* or in the *periosteum.* In adults the origin is usually periosteal, but in children almost exclusively medullary.

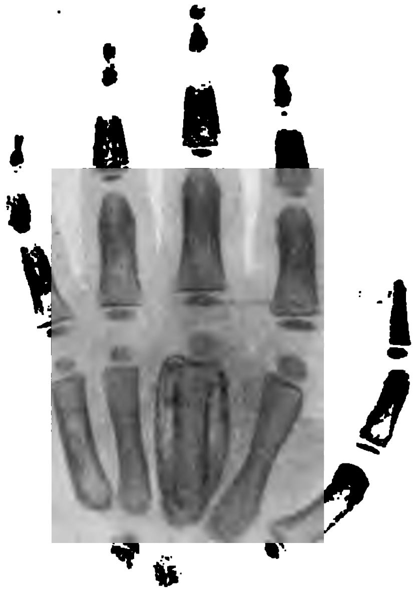


FIG. 423.—Tubercular dactylitis of periosteal origin.

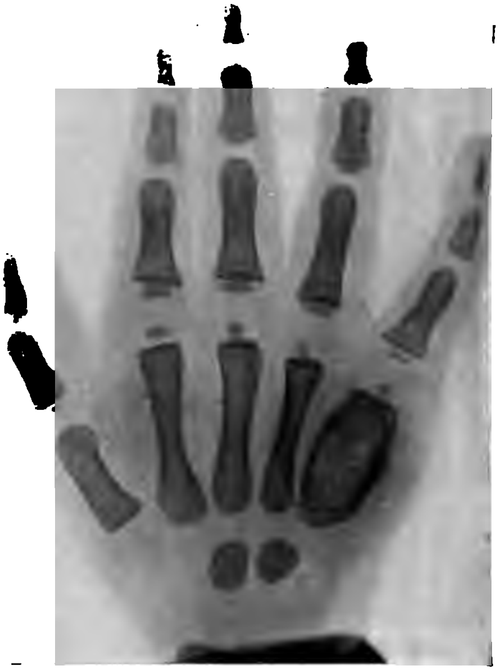


FIG. 424.—Tubercular dactylitis of medullary origin.

My impression is that children who are the subject of congenital syphilis also display the periosteal variety of tuberculosis more frequently. Whether this is actually a fact must, however, remain an open question.

The fate of the finger differs in the various forms of the disease. In tubercle arising from the medulla, ordinary tubercular dactylitis (fig. 424), the bone becomes more and more distended, or to put it more accurately, becomes destroyed internally and is replaced by new bone from the periosteum. In this condition, the periosteum may remain at least partially healthy. The spongy tissue becomes absorbed, or forms a sequestrum which is either expressed spontaneously or removed surgically. The phalanx becomes bent to one side, but practically the whole of it remains. In the periosteal form, however, the whole diaphysis gradually becomes deprived of its nutrition (fig. 423), necroses, and is expressed or removed surgically after prolonged suppuration. But as the periosteum itself is tubercular, no healthy involucrum is formed as in the case of staphyloccic osteomyelitis, but the finger becomes shortened by about the length of the diaphysis, after a few splinters of bone, formed from the periosteum, have been removed as sequestra.

The differential diagnosis between these two forms cannot be made clinically, at any rate as long as there is no fistula. It can, however, be made by a skiagram, upon which we must always base our treatment.

If there is a history of congenital syphilis or a positive serum reaction, the probability of syphilitic dactylitis is indicated. There is, however, no certain clinical symptom, and every case, which is not clearly one of tubercle, but in which there is some suspicion of syphilis, should have specific treatment before any other course is undertaken.

PART VII.

DISEASES OF THE LOWER EXTREMITIES.

CHAPTER XCIII.

DISLOCATIONS AND FRACTURES OF THE HIP.

THERE is no joint which presents so much difficulty to the beginner, and sometimes also to the experienced, as the hip, because of the inaccessibility of the articular ends both to sight and to palpation. All conclusions relating to the joint are, therefore, necessarily, based on indirect signs, and the hip thus places a severe tax on the diagnostic powers of the examiner.

The most striking symptom of all hip diseases is **limping**; and we shall therefore briefly consider the most important forms of this symptom. The simplest variety is that which follows *shortening*. In this condition, the body inclines towards the diseased side at every step; not because the limb gives way, but because it is too short. The leg is by no means spared, but still serves as a normal support. If the shortening is slight, the complete sole is planted down, but if it is extreme, only the toes reach the ground. The limp becomes noticeable in adults if the shortening exceeds 1½ cm. (⅝ in.).

The *paralytic limp*, in the widest sense of the term, is very similar. The limb is insufficiently supported, either because of muscular weakness or because of dislocation. The patient supports himself vigorously on the affected leg, evidently experiences no pain therein, but inclines with each step towards the diseased side, and then supports himself the more firmly on the healthy leg for the purpose of throwing the diseased leg forwards for the next step. If this variety of limping is due to congenital dislocation, the head of the thigh bone may be seen to move upwards, under the gluteal muscles, towards the pelvis. If the disease is bilateral, the gait is waddling, like that of a duck.

Painless stiffness of one hip produces quite a different kind of limp. The entire extremity, including the half of the pelvis, is moved forward as a whole, because a normal function of the other joints is not conceivable if *one* joint is stiff. But as there is no pain, the limb is not spared as a means of support, and the weight of the body is equally received by both legs. If the patient walks slowly, he is therefore able to render his disability less noticeable. The gluteal fold is obviously obliterated on the diseased side. A peculiar gait is produced by *bilateral stiffness of the extremities* as in cases of severe double coxa vara. The patient wearily moves forwards, first one half and then the other half of the pelvis alternately, and therewith the corresponding limb. The pelvis rotates around a vertical axis, and not around a sagittal axis, as in cases of bilateral dislocation.

In cases of *painful limping*, the movement of any joint is painful, and as all the joints of the lower limb are interdependent—for instance, a twisting movement of the foot is impossible without the participation of the knee and hip joints—the patient stiffens all the joints by muscular action, and avoids, as far as possible, putting any weight on the diseased limb, and inclines his body towards the healthy side. This latter circumstance distinguishes painful limping from limping due to painless rigidity, for otherwise most of the symptoms are common to both conditions, including especially the obliteration of the gluteal fold.

Sometimes the disturbance in gait is due to a combination of causes. In unilateral congenital dislocation, it depends upon shortening and upon laxity of the joint; in old hip disease, it depends upon shortening and upon stiffness.

After these preliminary remarks, we may now return to our main subject.

Doubt should rarely arise as to whether an injury to the hip-joint consists of a dislocation or a fracture. As a rule, such doubt indicates that the examination has not been conducted properly, or that the physical condition has not been correctly understood. In order not to fail in the appreciation of indispensable anatomical points, every practitioner should possess a skeleton, and he should consult it frequently.

A.—METHOD OF EXAMINATION.

We begin with *inspection*, and note the position and posture of the injured limb as well as the external visible injuries, extravasations of blood and swellings.

The experienced observer will often be able to make a diagnosis from the posture of the injured extremity, or at least will narrow down the possibilities to a very limited extent. Thus, if the injured person lies helpless and motionless, with his leg in a state of complete external rotation, he will at once think of a fracture below the neck of the femur. A flexed thigh, rotated internally and adducted, will suggest a dislocation. He will also notice whether the region of the hip, on the injured side, is drawn in, or whether it projects unnaturally. A roundish bulging in the inguinal region is not likely to escape him any more than the fact of the one patella being higher than the other —a condition which indicates shortening.

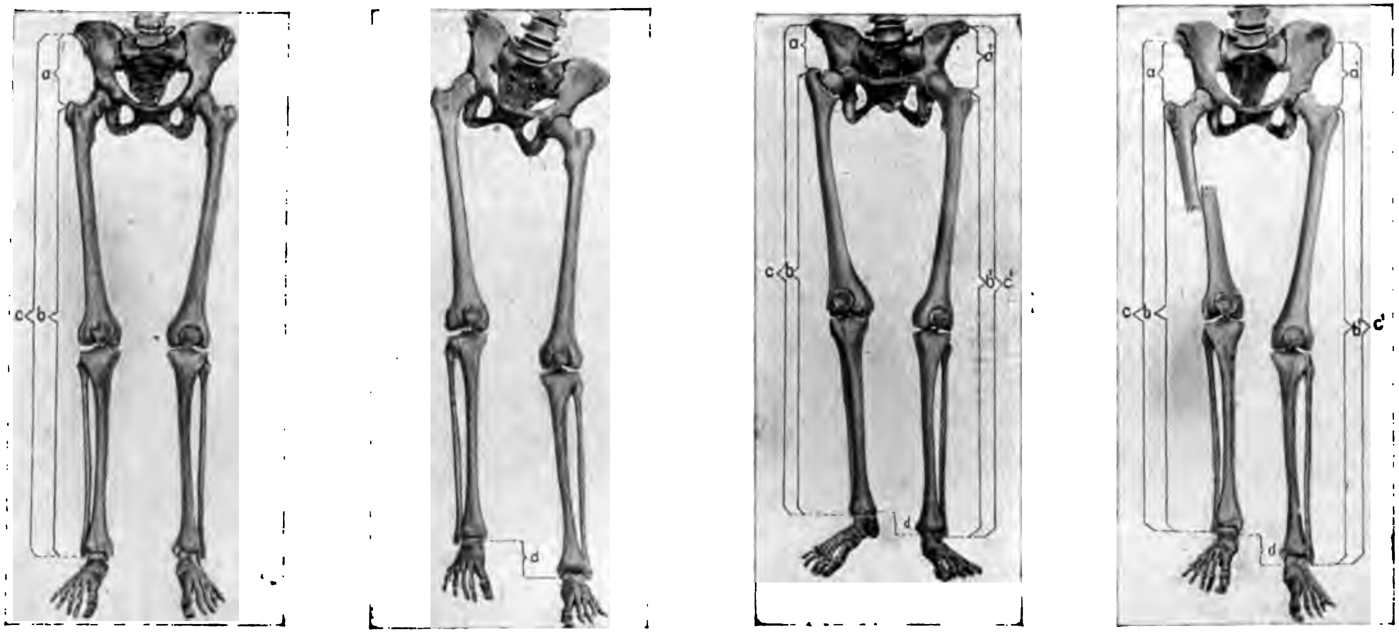
We next proceed to what is for the patient the least painful procedure of examination, the *measurement of the length* of the limb, and for this purpose, it is necessary to bring both limbs into exactly the same position, to avoid mistakes.

We measure on both sides the distance (1) between the anterior superior spine of the ilium and the tip of the external malleolus (*c* in fig. 425) and (2) the distance between the tip of the trochanter and the malleolus (*b*). In cases of extreme external rotation the first measurement is taken to the internal malleolus. If the measurements are equal on both sides, we may, as a rule, exclude any change in the bone, provided that the limb used as the standard for comparison has not been shortened by a previous accident. If one or other of these measurements, or both of them, be shortened, there is certainly either a dislocation or a fracture, unless the shortening be due to some previous injury or disease. If the distance between the anterior superior spine and malleolus (*c*) is alone shortened (supra-trochanteric shortening) the case is either one of dislocation or fracture of the neck of the femur. If both measurements *b* and *c* are shortened, the separation of continuity must be below the tip of the trochanter (infra-trochanteric shortening), and the case can only be one of fracture below the tip of the trochanter.

We next determine the relation of the trochanter to the pelvis, in order to control the measurements previously obtained. It is generally said that for this purpose the most important thing is to ascertain Roser-Nélaton's line. Unfortunately, it is very difficult to do this when the injury has been severe. It is better to make use of this line in cases of abnormal positions, of *non-traumatic* origin. In *recent injuries* we should adopt other measurements which are equally reliable and easier to carry out, and which do not demand any change of posture in the injured person. These measurements consist of Bryant's triangle and the trochanter—anterior superior spine—umbilicus line, which will be referred to in detail in the next section. The determination of Bryant's triangle is of special significance because the measurement of the horizontal base line of this triangle intimates to us the alteration in the position of the trochanter, and gives its level, and thus enables us, by comparing the two sides, to ascertain how far the trochanter has been displaced upwards.

If the trochanter is abnormally high, there is either a dislocation or a fracture of the neck of the femur. If it is in a normal position the injury is not associated with any displacement, or, if there be any, it must be below the trochanter.

In slight bending of the neck of the femur, or in fractures which are slightly impacted, the shortening may be so small in amount as to come within the ordinary limits of errors of measurement. In anterior dislocations the trochanter is not displaced upwards in any marked degree. But in these cases, its definite approximation to the mid-line of the body shows that some anatomical damage has been



(*a*)

Normal relations. a = Level of trochanter, as judged by length of horizontal line joining the ant. sup. spine to tip of trochanter (the patient being recumbent). b = Distance between trochanter and malleolus. c = Distance between ant. sup. spine and malleolus.

(*b*)

Apparent shortening. d, in consequence of adduction of right leg. (In the same posture pathological abduction of the left leg would produce apparent lengthening of the right.

(*c*)

Actual supra-trochanteric shortening. The difference between the levels of the malleoli, d, is equal to the difference between the distances from the ant. sup. spine to the malleolus on each side, c^1 and c, and is also equal to the difference between the lines joining the ant. sup. spine to the tip of the trochanter on each side, a^1 and a. The distances between trochanter and malleolus on each side are equal. $d = c^1 - c = a^1 - a$; $b = b^1$. This condition is found in fractures of the neck of the femur proper, in coxa vara, and congenital dislocation of hip.

(*d*)

Actual infra-trochanteric shortening. Difference in level of malleoli, d, equal to differences in distances between ant. sup. spine and malleolus, and also equal to difference in distances between trochanter and malleolus. Level of trachanters, same on both sides. $d = c^1 - c = b^1 - b$; $a = a^1$. This condition is found in all fractures through or below the trochanter, and also in all fractures lower down the limb.

FIG. 425.—Length measurements of lower limb.

sustained. We also find a certain amount of approximation to the middle line in impacted fractures of the neck of the femur. But, as we shall see later on, there are other definite signs which distinguish these fractures from anterior dislocations.

After ascertaining the measurements of the limb, we must determine the range of *active movements.*

For this purpose, the patient must lie flat in bed, undressed, and endeavour to raise the injured limb in the position of extension. If he can do this without hesitation, although there may be pain, there is certainly neither dislocation nor fracture. A sprain, or a contusion is the most likely thing. If he flexes the thigh with difficulty, without, however, raising the heel from the bed, the case may probably be one of impacted fracture. We then ask the patient to perform movements of rotation. If external and internal rotation are perfectly free, there can be no severe injury present. But if there is a definite limitation of active internal rotation, whereas outward rotation appears to be normal, or even exceeds the normal in extent, the case is probably one of impacted fracture of the neck of the femur. If no active movements of rotation are possible, and at the same time the limb is turned outwards, there is probably a fracture below the trochanter.

We cannot elicit much information from abduction and adduction movements, because the raising of the whole leg is necessary to perform them. But the same remarks apply to these movements as to rotatory movements.

We now proceed to *passive movements,* which must perhaps be carried out under anæsthesia, if a skiagram cannot be obtained. It is obvious that the manipulations must be performed with the greatest care, lest a useful impaction becomes separated in the process. It is always wrong to separate an impaction, and the procedure is only excusable if the rotation of the shaft is abnormal, or the adduction too pronounced.

If passive movements are free, or only slightly hampered on internal rotation, the case is certainly one of *fracture,* assuming that a severe injury has been sustained. If the movements exceed the normal range in certain directions, and encounter a spring-like insurmountable resistance in the opposite directions, the case is certainly one of *dislocation.*

This rule is subject to the slight limitation that there is some diminution of internal rotation on passive movement, in cases of impacted fracture of the neck of the femur, and, exceptionally, there is also diminution of external rotation.

Finally, we proceed to *palpation.* In dislocations, the head of the femur can be felt in an abnormal position, and in fractures we can recognize thickening of the trochanteric mass and strikingly abnormal protuberance of bone. The examination of active and passive movements, and the palpation afford the opportunity of noting the degree and the localization of the *pain.*

We have now arrived at the stage wherein we may apply the results of our examination to the formation of a diagnosis.

B.—DIAGNOSIS OF THE VARIOUS FORMS OF INJURY.

(1) DISLOCATIONS.

Having diagnosed a **dislocation,** it remains to determine its variety :—

(*a*) If the thigh is rotated *inwards*, a simple mechanical consideration will show that the head of the femur must have gone *backwards*, and therefore the case is one of *posterior dislocation*. In ordinary dislocations the Y-shaped ligament is not ruptured, and this structure maintains the thigh in the position of flexion and adduction. If the three characteristic anomalies of posture—internal rotation, adduction, and flexion—are not very pronounced, we may conclude that the head of the femur has escaped in an upward direction, towards the iliac fossa (fig. 426) **iliac dislocation.** If, however, the abnormal posture is very pronounced, we assume that the head has escaped in a backward and downward direction—**a sciatic dislocation** (fig. 427*a*). In the former case the rent in the capsule is at the back and above; in the latter case it is at the back, or behind and below. We may summarize the differences into a definite law as follows: *An iliac dislocation exists if the patient, on lying down, is able to conceal the flexion by means of compensating lordosis of the lumbar spine, and bring his two legs into an approximately parallel position, and if, on standing up, he is able to touch the ground with his toes. On the other hand, a sciatic dislocation is present, if the most extreme lumbar lordosis is unable to abolish the flexion, if the patient, on lying down, places the thigh of the dislocated leg on the healthy thigh, and if he is unable to touch the ground with his toes on standing up.*

It sometimes happens that our examination points to an iliac dislocation, but we are told that the abnormality of position was much more striking immediately after the accident. This suggests that the dislocation was originally of the sciatic variety, but that, as a result of the weight of the limb or of the attempts at reduction, the head of the femur has glided upwards and reached the position of an iliac dislocation.

It is absolutely impossible to mistake posterior dislocation for any other injury of the hip. Impacted fractures of the neck of the femur with internal rotation do certainly occur, but very rarely. But in such cases the freedom of abduction and the absence of the head of the bone from an abnormal situation show that no dislocation exists.

If a *posterior dislocation is complicated by other injuries*, greater difficulties of diagnosis occur. If the injury has resulted from some great violence, the Y-shaped ligament may occasionally be torn and then a regular dislocation be converted into an irregular one. The limitations of movement which ordinarily indicate a dislocation would not be present in such a case, but, on the other hand, the head of the femur would be more accessible to palpation, and thus afford evidence of the dislocation. In some typical cases of posterior dislocation, grating and crepitus are heard, which lead to errors in diagnosis and suggest fracture of the neck of the femur. As a matter of fact, a *portion of the margin of the acetabulum* has been torn off in these cases. If the piece of acetabulum broken off is large, the displaced

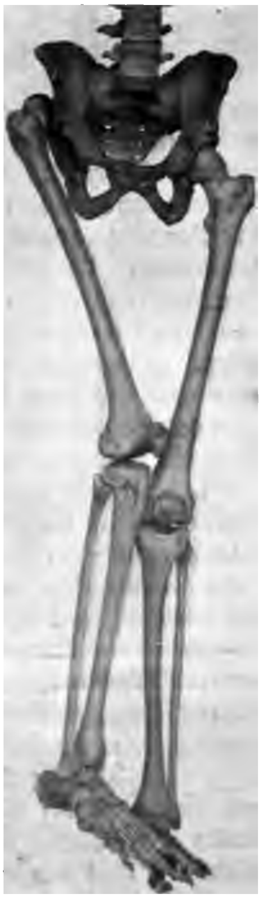


FIG. 426.--Iliac dislocation.

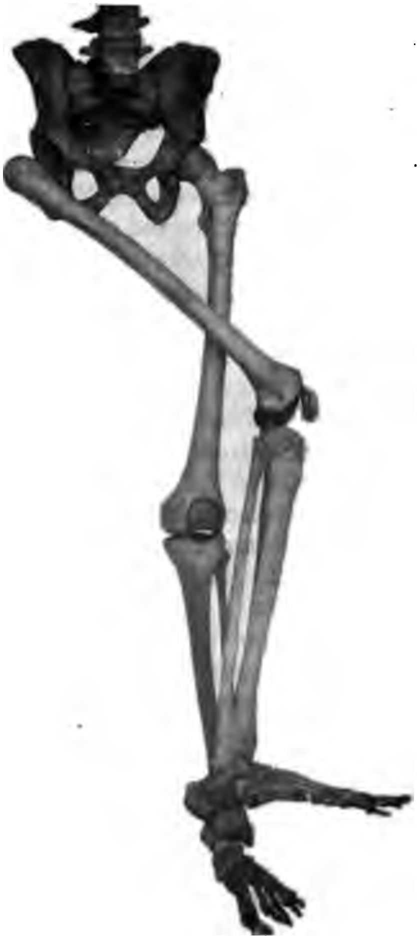


FIG. 427*a*.—Sciatic dislocation.

head will not be retained in any of the typical positions of dislocation. In such a case the diagnosis from subcapital fracture can only be made by means of a skiagram (fig. 428). A *combination of dislocation with fracture of the neck of the femur* is very difficult to diagnose, because it presents a very unusual picture. The diagnosis can only be arrived at by demonstrating that the head of the bone does not follow the limb on rotation, and that it is in an abnormal position.

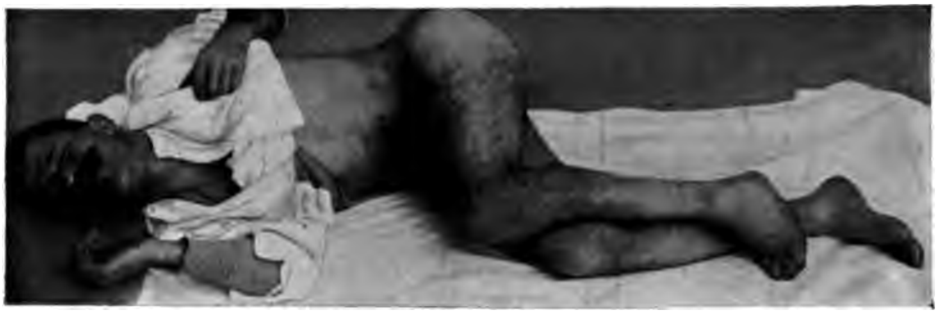


FIG. 427*b*.—Sciatic dislocation.

(*b*) If there is limitation of movement indicating dislocation, and the limb is turned *outwards*, we must conclude that the head of the bone has escaped *forwards*, and that, therefore, an *anterior dislocation exists*. We have already seen that the approximation of the trochanter to the middle line is another indication of this injury. As the **Y**-shaped ligament remains intact a more or less considerable abduction results. If this abduction is not very pronounced, and the injured limb can be placed parallel to the sound one, and is also in a position of extension, the dislocation is *forwards and upwards*. It is termed **ilio-pectineal dislocation** or **pubic dislocation** in accordance with the situation in which the head of the bone is found (fig. 429). The fact that the head of the bone can be felt on the horizontal ramus of the pubis in the inguinal region furnishes a clinching demonstration

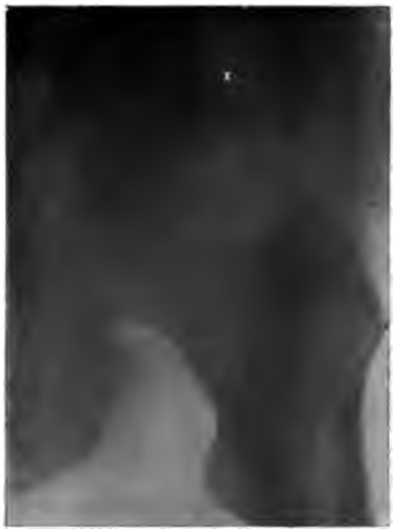


FIG. 428.—Portion of upper margin of acetabulum detached.

of the accuracy of the diagnosis. The slight abduction gives the limb the appearance of some lengthening, and when the sound limb is adducted close to the injured one the sound limb looks shortened and the injured limb lengthened. If both limbs are, however, measured in the same position, some shortening of the distance between the anterior superior spine and the malleolus will always be found.

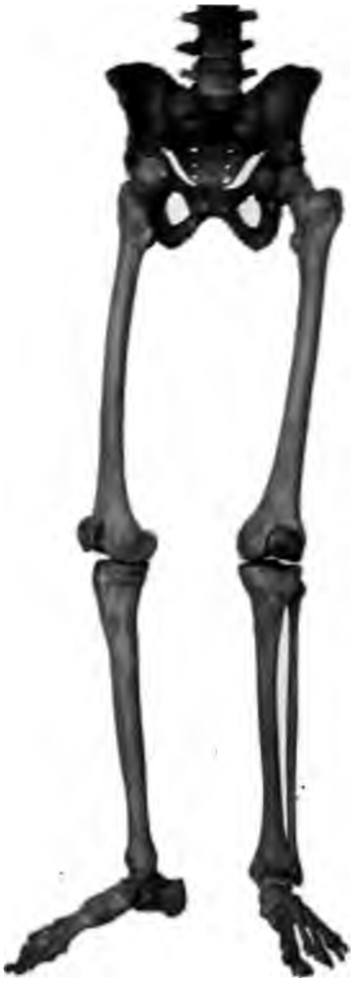


FIG. 429. Ilio-pectineal dislocation.

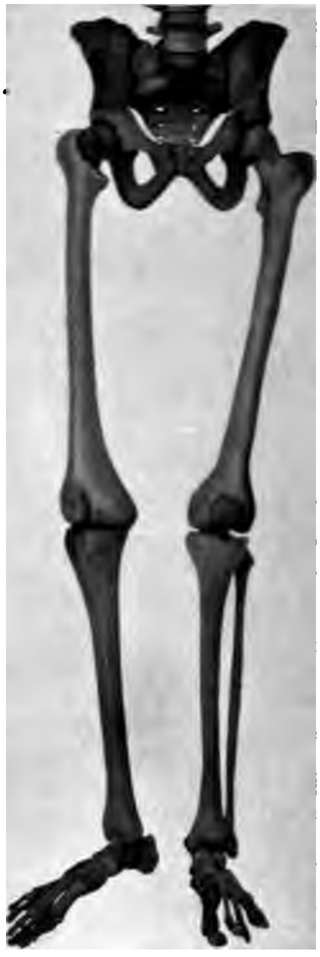


FIG. 430.—Inter-trochanteric fracture.

In this dislocation the femoral artery can be felt internal to the head of the femur, and, indeed, the artery may be lifted up by it.

Neuralgic pains and disturbances of sensation in the area of the anterior crural nerve indicate that this nerve has been dragged upon.

If the abduction is much more definitely pronounced, and is associated with flexion, and the head of the bone is not found on the horizontal ramus of the pubis, we make the diagnosis of **obturator dislocation.** In well-developed muscular persons the head of the bone cannot easily be felt. In confirmation thereof the region of the trochanter will not only be flattened out, as in suprapubic dislocation, but will also be drawn in. Finally, we shall infer, from radiating pains and paræsthesia on the inner side of the thigh, that the obturator nerve has been compressed.

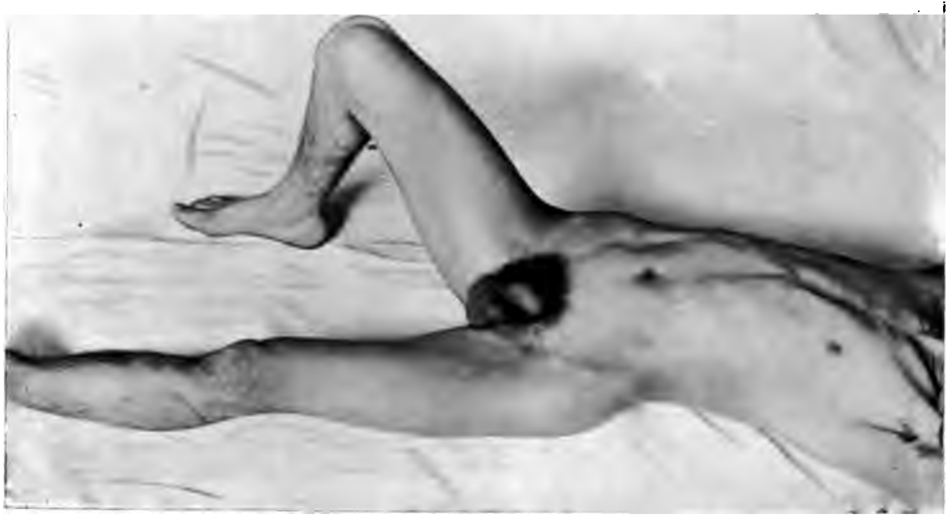


FIG. 431.—Obturator dislocation.

If a case presents, with outward rotation, an increase of the flexion to a right angle, we may diagnose the very rare dislocation towards the perinæum, i.e., *perineal dislocation.*

We may now consider what mistakes may arise in connection with anterior dislocations. Supposing the external rotation in a case of suprapubic dislocation would, for a moment, suggest fracture of the neck of the femur (*cf.* figs. 429 and 430), the presence of the head of the bone in an abnormal position should at once establish the diagnosis of dislocation with so much certainty that no other differential signs would be required. Obturator dislocation is distinguished from fracture of the neck of the femur by abduction of the limb, by considerable flexion, and also by indrawing of the trochanter region (fig. 431). This last sign is only equally pronounced in fracture of the pelvis when the head of the femur is driven into the pelvic cavity—the so-called *central dislocation.*

Perineal dislocation, with the very unusual posture arising therefrom, cannot be mistakén for any fracture.

(*c*) It is necessary to mention finally the very rare dislocations, *upwards* and *downwards*.

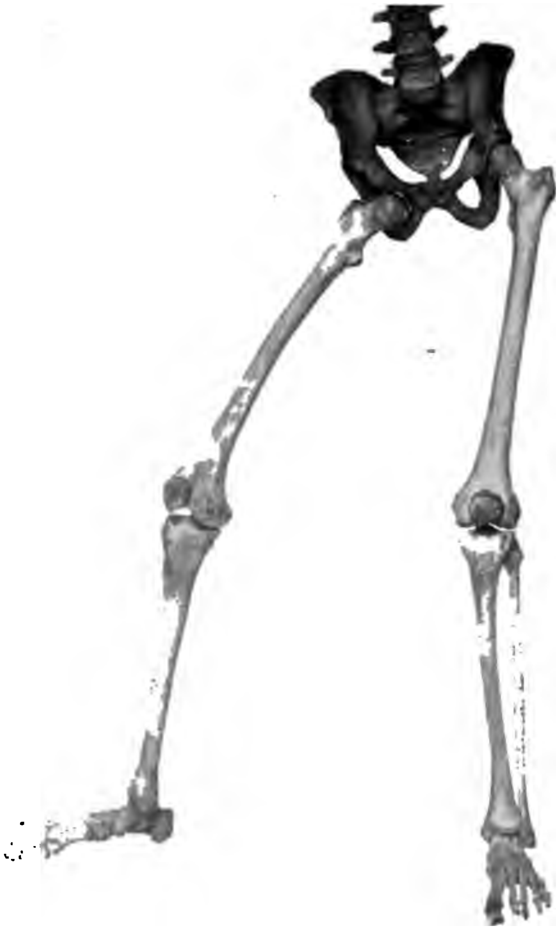


Fig. 432.—Obturator dislocation.

Supracotyloid dislocation resembles a suprapubic dislocation, forwards and upwards, except that the head of the bone is felt immediately below the anterior superior spine. This form is easily recognized. **Infracotyloid dislocation,** i.e., downward dislocation, is recognized by the flexion of the thigh to a right angle, with slight external rotation and abduction. It approximates in signs to an obturator dislocation.

(2) CONTUSIONS, SPRAINS, FRACTURES.

If the passive movements in the foregoing examination have been free, and therefore the possibility of dislocation is excluded, we have to decide between **contusion, sprain** and **fracture.**

Some indications will already have been furnished by the *cause.* A contusion arises from a direct injury to the hip. But, in the absence of such a cause, a sprain is the most likely diagnosis, as, for instance, when an elderly man collapses after an excessive movement of external rotation. This injury would not, however, completely abolish the supporting function of the limb. If the patient makes fruitless attempts to stand and to walk, and finally allows himself to be carried home and put to bed, we may assume the presence of a fracture. But if he walks home on foot, the case is either one of contusion or sprain, in accordance with the form of the injury.

There is, however, one important exception to this rule, which not so much concerns the fact that sometimes contusions and sprains cause very grave disturbances of function, as that certain fractures are attended by very little derangement. It happens, especially in the case of impacted fractures, and even in separation of the epiphyses in young people, that the patient is often able to walk home. If the practitioner relies upon this circumstance to exclude a fracture, and neglects a more careful examination, both he and the patient may be confronted, in the course of a few weeks, with the unpleasant surprise of a healed fracture, with shortening and external rotation, i.e., a *traumatic coxa vara;* unless the fracture has, in the meantime, been diagnosed by another practitioner. I have seen a female patient aged 81, who was able to walk about after a sub-capital fracture.

If the patient lies extended on his bed, the position of the limb affords certain indications. A normal position is in favour of a simple contusion, as most fractures of the neck of the femur are associated with external rotation.

We have already mentioned the rare cases of impaction in internal rotation. This possibility must always be thought of, lest a dislocation be wrongly diagnosed.

We must also consider the amount of spontaneous pain and the painfulness of the joint, when it is interfered with. As Kocher points out, the spontaneous pain in contusion may be disproportionately great, despite slight loss of power, whereas in fracture it may be very slight, despite complete loss of power. On the other hand, the pain caused by pressure exerted in the axis of the femur is slight or completely absent in contusion, whereas it is always present in cases of recent fracture. Thus we arrive at the following rule :—

If a person suffers a severe loss of power in consequence of a direct or indirect, seemingly insignificant injury to the hip, a fracture is highly probable. The greater the contrast between spontaneous pain and the extent of functional disturbance, the more likely it is that a fracture has taken place. If the supporting function of the limb is abolished, although the spontaneous pain is only slight, there is certainly a fracture present.

In these circumstances, there is no difficulty in bringing forward direct evidence of the fracture. It is certain that a complete fracture exists if there is *shortening* which cannot be attributed to a previous injury, or if the trochanter is above its normal position, or if there is an approximation of the femur to the pelvis.

Partial fractures of the trochanter, detachments or abrasions of small pieces thereof, can only be distinguished from simple sprains by means of a skiagram, because of the great effusion of blood into the soft parts.

Complete detachment of the great trochanter, which is rare, may be diagnosed by the loss of power of support in the hip, despite the normal condition of the joint, and by the demonstration on palpation of the upwardly displaced fragment.

Detachment of the small trochanter, which is still more rare, may be recognized, according to Ludloff, by the fact that the patient cannot raise his limb when in a sitting posture.

We must not base the diagnosis of a bone lesion merely on the presence of crepitus. There are cases in which, independently of any injury, the anterior margin of the tendon of the gluteus maximus, or the ileo-tibial band of the fascia lata, glides over the trochanter with palpable and audible crepitus, when the muscle contracts powerfully. This has been termed in French "*hanche à ressort*," but the German expression, "*schnappende Hüfte*," is more expressive.

Having decided that a complete fracture exists, we must next determine where it is situated, and whether it is loose or impacted. We cannot rely on skiagraphy, even as much as in elbow or shoulder cases, because the conveyance of a patient with an injured hip to an X-ray institute is generally quite impracticable. But, nevertheless, a correct diagnosis is most important for prognosis and treatment.

We may here briefly review the various forms of fracture. It was formerly the custom to distinguish between *intracapsular* and *extracapsular fractures*. But as the capsule extends farther towards the trochanteric region on the anterior surface than it does on the posterior surface, and as also the line of fracture is often irregular, it follows that numerous fractures of the neck of the femur are of a mixed variety, partially extra- and partially intra-capsular. We, therefore, adopt Kocher's classification and divide these fractures according to their position, regardless of their relations to the capsule.

One line of fracture (fig. 433, 1) lies at the border between the head and the neck. This variety is termed by Kocher, *fractura subcapitalis*, and if pure, is always intracapsular. Another line of fracture lies along the neck itself, where it merges with the trochanter mass, that is to say in the region of the intertrochanteric line (fig. 433, 2)—*fractura intertrochanterica*. This fracture is partially intra- and partially extra-capsular. The *pertrochanteric fracture*, the line of which traverses the trochanter mass, lies close to the line of the fracture of the neck of the femur. It generally runs obliquely, from outwards and upwards in front, to downwards and inwards behind (fig. 433, 3): this fracture is more common than the pure intertrochanteric variety. The *subtrochanteric fracture*, which traverses the bone below the lesser trochanter, belongs to the fractures of the shaft, but it is more practical to discuss it with the fractures of the neck of the femur (fig. 433, 4 and 5).

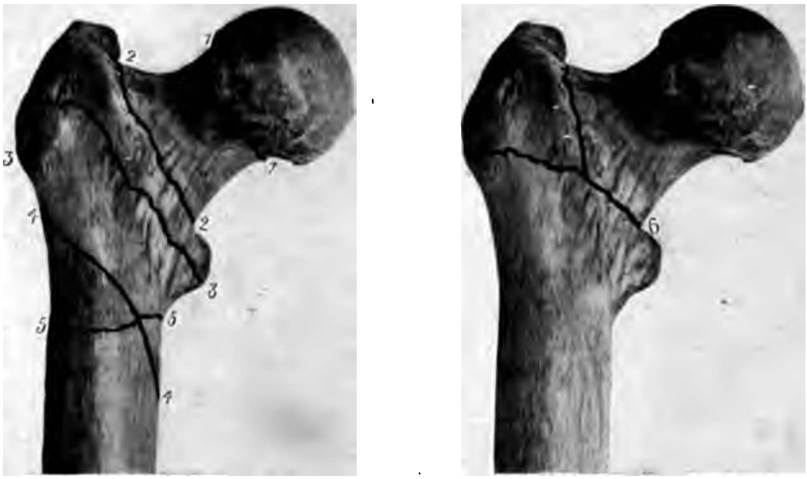


FIG. 433.—*a*—*b*, Typical lines of direction of fractures at the neck of the femur.

a
1. Fractura subcapitalis.
2. „ intertrochanterica.
3. „ pertrochanterica.
4. „ subtrochanterica (oblique variety). Rotation fracture.
5. „ Ditto (transverse variety.)

b
6 Y-shaped fracture.

Intertrochanteric and pertrochanteric fractures are not usually pure in form. The former is very often associated with a fracture in the trochanter mass—indeed recent autopsies show that this is probably the rule—so that really a Y-shaped fracture results (fig. 433, 6). Pertrochanteric fractures may also be combined with intertrochanteric, as well as with subtrochanteric fractures.

Impaction may occur in all forms, except in a pure subtrochanteric fracture.

How much of this can be recognized with certainty in the living subject without X-rays ?

(*a*) In the first place we must decide whether a **pertrochanteric** or **subtrochanteric fracture** is present or not. If the fracture involves the great trochanter below the insertion of the Gluteus medius active internal rotation is impossible, and the limb falls into a position of eversion by its own weight and by the action of the external rotators (Quadratus femoris and often the ilio-psoas). This eversion varies inversely with the impaction, the latter being less, the lower the fracture.

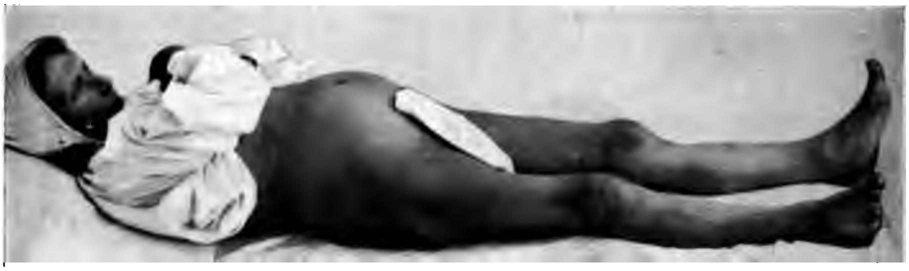


FIG. 434.—Intertrochanteric fracture. Incomplete external rotation. Shortening.

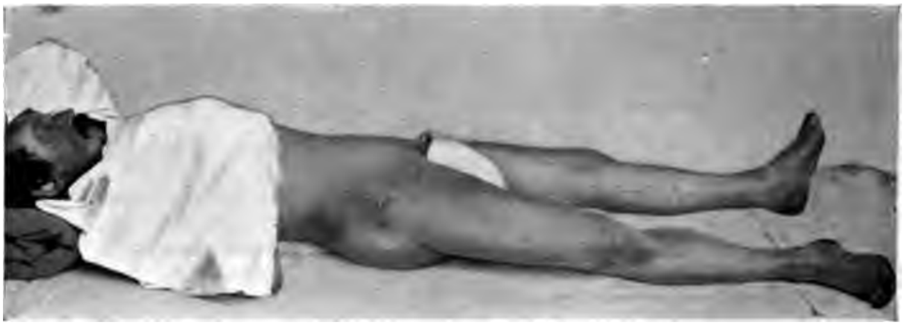


FIG. 435.—Subtrochanteric fracture. Complete external rotation. Shortening.

The measurements of the limb and the condition of the trochanter are decisive. If there be shortening of the distance between the anterior superior spine and the malleolus, and of the distance between the trochanter and the malleolus, with the trochanter in a normal position, the fracture must be below the trochanter. If, on the other hand, only the distance between the anterior superior spine and malleolus is shortened, and the trochanter is abnormally high in position, the fracture cannot be below the trochanter (see fig. 425). If the intense swelling of the soft parts or some old shortening render the results of measurement indecisive, the case may be elucidated by the *behaviour of the trochanter on rotation of the thigh*. If the tip of the trochanter revolves with the rotation, the fracture is above it; if it does not revolve, the fracture is below it. An impacted pertrochanteric fracture is often recognizable by the presence of a double prominence instead of the great trochanter, further by the tenderness over the trochanter itself, and often by the involvement of the thigh in the swelling.

It is further necessary to distinguish a sub-trochanteric from a pertrochanteric fracture. The differentiating features of the former are great eversion, complete helplessness of the limb and absence of definite enlargement of the trochanter. The pain on pressure is mainly felt below the trochanter mass, and there is a hæmatoma which extends far down the limb.

In a typical pertrochanteric fracture, wherein the plane of the fracture runs from the front downwards and backwards, it is very often possible to feel the sharp edge of the *lower* fragment, which is displaced upwards and forwards, as Kocher has pointed out. On the other hand, in a pure subtrochanteric transverse fracture, the lower end of the *upper* fragment, which is kept flexed by the ileo-psoas, can be felt beneath the muscles.

(*b*) If we have excluded both a per- and sub-trochanteric fracture, and therefore limited the diagnosis to **fracture of the neck of the femur**, we still have to distinguish between a *subcapital* and *intertrochanteric fracture*, and also to decide whether it is *impacted* or *non-impacted*.

We first endeavour to localize the seat of maximum *pain on pressure*. If it is beneath the middle of Poupart's ligament in the neighbourhood of the head of the femur, we may conclude that the fracture is subcapital. If the site of maximum pain on pressure is behind and internal to the trochanter mass, we may assume that the fracture is intertrochanteric. At the same time, we notice whether there is any marked *swelling*. If there be any in the neighbourhood of the trochanter mass it is clear that an intertrochanteric fracture must be present. The examination of the trochanter for pain on pressure is made by the tips of the second, third and fourth fingers.

The question of impaction is settled by the power of spontaneous movement, and the examination for unnatural mobility. The more of the former and the less of the latter, the more likely is the fracture to be impacted, and vice versa. Crepitus obviously contra-indicates impaction. Experience shows that the nearer the fracture approaches the trochanter mass, either from below or from above, the more likely is it to be impacted.

In some cases the *etiology* assists in the forming of a diagnosis, but very often the patient himself does not know the details of the accident, and his statements are quite indefinite and useless. If he has fallen on his feet and the blow has thus been imparted from below, a subcapital fracture is the most likely result. A fall on the region of the trochanter is the most frequent cause of intertrochanteric fracture. Extreme external rotation is just as likely to be due to pertrochanteric or to subtrochanteric fracture as to subcapital fracture, but does not suggest intertrochanteric fracture.

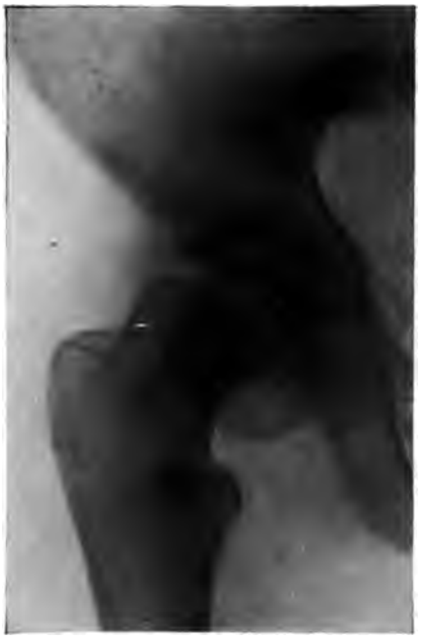


FIG. 436.—Separation of epiphysis in a girl aged 14.

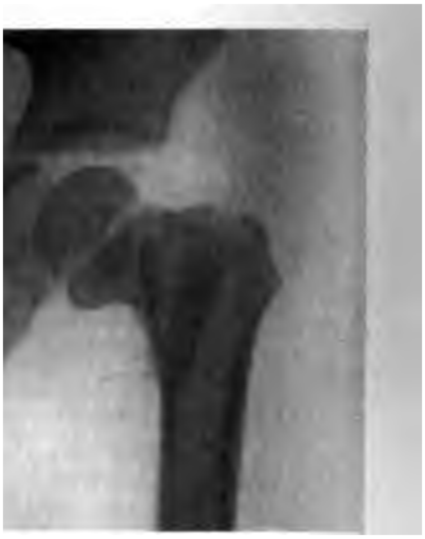


FIG. 437.—Per-trochanteric in a girl aged 4 years.

So far we have purposely not laid any stress on the *age* of the patient. Too much importance has hitherto been attached to the statement that fracture of the neck of the femur is the prerogative of old people. There is no doubt that old age predisposes to it, but fracture of the neck of the femur is by no means a rarity in young persons.

The patients, after a slight injury, remain in bed for a few days, sometimes even for two or three weeks, with the diagnosis of contusion of the hip, or with no diagnosis at all, and then begin to resume their occupation. After a few months have elapsed, they consult their doctor because of pains in the hip and slight lameness. Examination then reveals a shortening of 1 to 2 cm., a corresponding elevation of the level of the trochanter, diminution of the power of abduction and possibly some slight external rotation. This, of course, establishes the diagnosis of *coxa vara*, and a careful investigation of the history may be required in order to detect the traumatic origin of the malady.

It may be quite impossible to distinguish between traumatic and spontaneous coxa vara, and even a skiagram may not always enable us to come to a conclusion, as illustrated in fig. 456*a*.

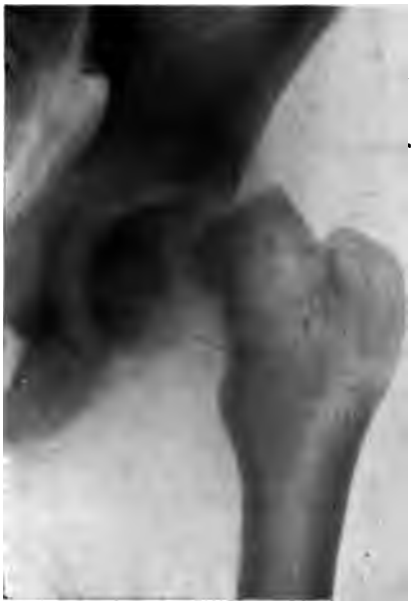


FIG. 438.—Subcapital fracture.

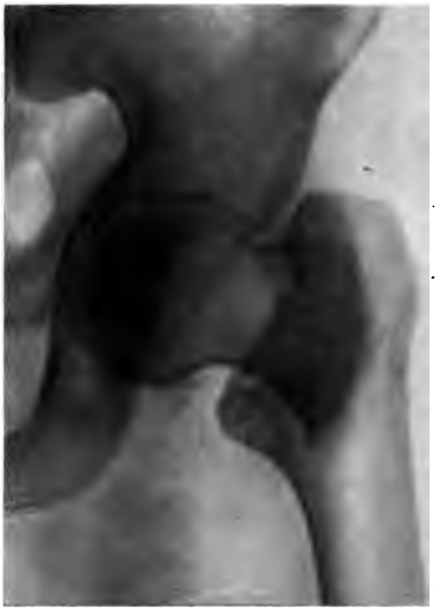


FIG. 439.—Intertrochanteric fracture.

The matter is of practical importance in regard to accident insurance. As far as our present experience goes, it would appear that the accident should always be held responsible, if an injury has really been proved, and if no hip symptoms existed previously. The other hip should always be examined and a skiagram made. If it shows signs of commencing coxa vara the accident can only be credited as an aggravating factor.

Much controversy has raged as to whether fractures during the period of growth are **separations of the epiphysis** or not.

Experience shows that loss of continuity of the bone occurs frequently in young people, in the region of the epiphyseal cartilage, but that the separated surface is not always in the epiphyseal line in any limited extent. Children however are liable to genuine inter- and per-trochanteric fractures (fig. 437).

Can a fracture of the neck of the femur be *mistaken for any other injury*, apart from dislocation, contusion, and sprain, which have already been referred to?

Fractures of the shaft of the femur do not come into serious consideration, because the seat of the injury is very clearly evident on palpation and on testing for abnormal mobility. But, on the other hand, in very severe injuries, such as those sustained in the collapse of a building, a fracture of the neck of the femur may be associated with fracture of the shaft, and the diagnosis thus be rendered difficult.

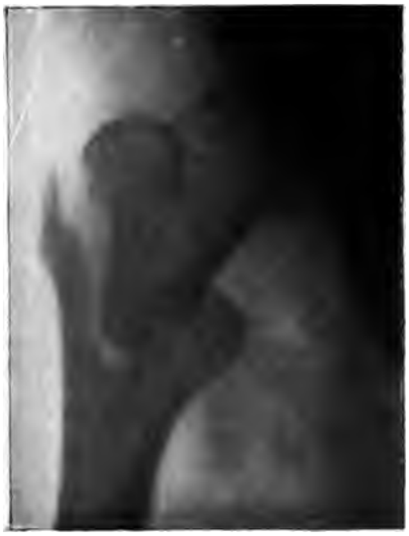


FIG. 440.—Per-trochanteric fracture.

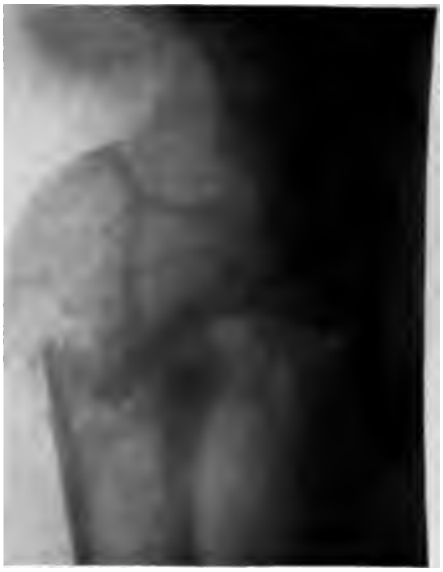


FIG. 441.—Subtrochanteric fracture.

It is, however, quite conceivable that a **fracture of the pelvis** may be mistaken for a fracture of the neck of the femur.

If there is a striking limitation of active movements, especially of flexion, although the measurements and the passive mobility are normal, we should think of a *fracture of the ring of the pelvis, unaccompanied by displacement*. This would be confirmed by pain on pressure on the pelvic fossa, by pressing the two pubic bones away from one another, and by force applied in the axis of the femur. We should endeavour to determine the exact course of the fracture by palpating the accessible portions of the pelvis, more especially the iliac crest, the pubis (both internally and externally), and the sacrum.

If, in addition to the loss of power just described, there is also shortening, which is evident to the eye, but which cannot be demonstrated by the tape measure, we must conclude that the entire portion of the pelvis, embracing the acetabulum and the anterior superior spine, has been separated from its connections through a *double fracture of the pelvic ring*, and has been at the same time displaced upwards (double vertical fracture of Malgaigne). The limb is usually everted in these cases, just as in a fracture of the femur.

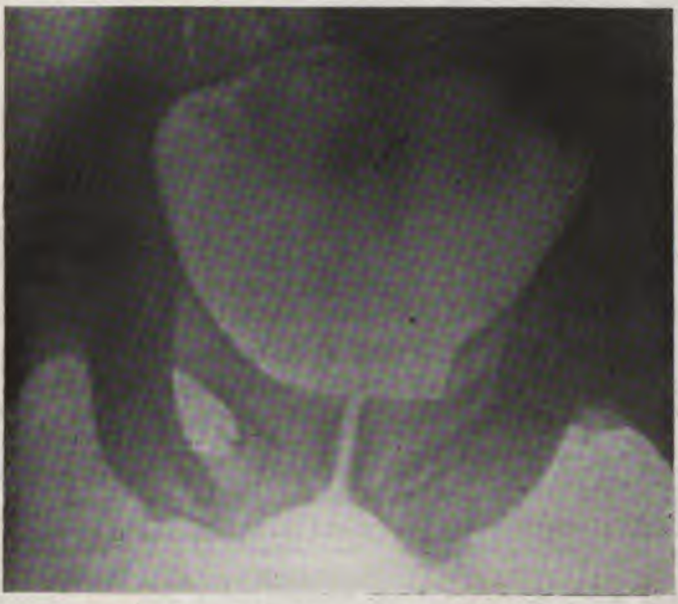


FIG. 442.- Bilateral fracture of the pubic bone.

The limb may appear to be lengthened, although the measurements are normal, in cases wherein the one half of the pelvis is displaced downwards in relation to the other half, as a result of a complete fracture of the pubis and a simultaneous loosening of the sacro-iliac articulation.

If the distance between the anterior superior spine and the malleolus is lengthened, without simultaneous lengthening of the distance between the trochanter and the malleolus, and without evident lengthening of the whole limb, and if at the same time passive movements are normal, and the supporting power is retained, the case can only be one of fracture of that portion of the pelvic fossa which includes the anterior superior spine (Duverney's fracture).

Pressure on the pelvic fossa may elicit pronounced pain, and possibly crepitus in confirmation of this assumption. There is no pain on pressure in the axis of the femur.

If the limb is shortened and fixed in a position of eversion, but the head of the bone cannot be felt to be displaced forwards as in an ileo-pubic dislocation, the acetabulum must be shattered, and the head of the bone driven into the pelvis. Rectal examination will confirm this diagnosis of *central dislocation*, which is a very rare injury.

If the hip-joint is quite free, but pain is caused in the *sacro-iliac articulation* by pressure or traction, the cause must either be a *sprain* or a *fissure* in the vicinity of the articulation. If nothing can be detected on palpation, the diagnosis must be made by means of the X-rays. Circumscribed pain on pressure over the inguinal region indicates fracture of the pubic bone. The diagnosis is confirmed if we find localized pain on pressure over the descending ramus of the pubis, as felt through the perinæum or *per rectum*.

In all cases of pelvic fracture attention must be paid to the *condition of the urinary tract*, for, as already stated, the urethra may be injured in various ways, and the bladder may be impaled by a splinter of bone. It is sometimes the urinary symptoms which draw attention to an overlooked fracture of the pelvis.

The following table will enable the beginner to appreciate the symptomology of injuries to the hip with greater facility :—

Active and passive movements free in all directions, but partially painful; power of support retained; no shortening	Direct trauma (Ecchymoses)		**Contusion (rarely an impacted fracture with slight displacement).**
	Indirect trauma	Active flexion quite free	**Sprain.**
		Active flexion impossible in the sitting posture	**Detatchment of lesser trochanter.**
Active and passive movement abnormally free in certain directions, and restrained in others; power of support almost always lost, at any rate at first	Limb inverted, adducted and flexed; head to be felt on the pelvis	The foot reaches the ground on standing (with the other limb extended), or at least the heel of the other foot	**Iliac dislocation.**
		The foot does not reach the ground	**Sciatic dislocation.**
	Limb everted and somewhat abducted	Limb extended; abduction slight; head visible and palpable over the pubic crest	**Ileo-pectineal and pubic dislocation.**
		Head not palpable there	**Central dislocation.**
		Limb semi-flexed; pronounced abduction; head not distinctly palpable	**Obturator dislocation.**
Active movements entirely lost, or almost so; passive movements free in all directions, or somewhat limited on internal rotation; shortening (sometimes very slight)	Level of trochanter normal; distance between ant. sup. spine and distance between trochanter and malleolus shortened (infratrochanteric shortening); tip of trochanter does not follow on rotation; limb completely everted	Trochanter mass painful on pressure up to the top, thickened; upper end of lower fragment often to be felt near tip of trochanter.	**Non-impacted per-trochanteric fracture.**
		Pain on pressure below the trochanter mass; upper fragment often to be felt under the skin in flexion	**Subtrochanteric fracture.**

Active movements entirely lost, or almost so; passive movements free in all directions, or somewhat limited on internal rotation; shortening (sometimes very slight)	Level of trochanter very high; distance between ant. sup. spine and malleolus shortened; distance between trochanter and malleolus normal (supratrochanteric shortening); tip of trochanter follows on rotation; limb usually in semi-eversion	Slight active movements present; sometimes even some power of support; no crepitus; femur not movable on pelvis; trochanter mass and tip of trochanter clearly palpable, and not painful on pressure externally	**Impacted intertrochanteric or Subcapital fracture** (distinction often impossible clinically without X-rays).
		Trochanter mass widened, painful on pressure externally; two protuberances to be felt on tip of trochanter	**Impacted per-trochanteric fracture,** or mixed form with **impacted intertrochanteric fracture.**
		Active mobility slight; no power of support; femur movable on pelvis with crepitus; pain on pressure, especially under Poupart's ligament, and not in the trochanter region; trochanter describes a curve on rotation	**Free Subcapital fracture** or **separation of epiphysis.**
		Active mobility and power of support *nil*; femur movable on pelvis with crepitus; pain on pressure on inner side of trochanter (as felt from behind); latter revolves on itself (i.e., in its long axis)	**Free intertrochanteric fracture.**
	Distance between ant. sup. spine and malleolus normal; passive movements free; active movements also free, but some looseness of support on standing	Localized pain on pressure over great trochanter	**Fracture of great trochanter.**
	Distance between trochanter and malleous normal; distance between ant. sup. spine and malleolus increased; function of hip normal	Compression of pelvic fossa painful also, but apparent shortening	**Fracture of pelvic ring** without displacement. **Double vertical fracture.** **Fracture of pelvic fossa.**

CHAPTER XCIV.

NON-TRAUMATIC DEFORMITIES AT THE HIP-JOINT, (CONGENITAL DISLOCATION OF THE HIP AND COXA VARA).

Coxa vara and congenital dislocation of the hip are obviously two very different diseases, and yet they are sometimes mistaken for one another. Even experienced observers are in doubt in certain cases. The difficulty is easily solved by a skiagram, but the apparatus for this purpose is not always available. We must, therefore, endeavour to establish the diagnosis without this aid.

A.—CONGENITAL DISLOCATION OF THE HIP.

In congenital dislocation of the hip, the head of the bone is no longer in the acetabulum, but is either above or below it. The first and inevitable result of this change in position is a *shortening of the distance between the anterior superior spine and the malleolus* and an elevation in the *level of the trochanter.* The shortening amounts to about 2 cm. even in little children.

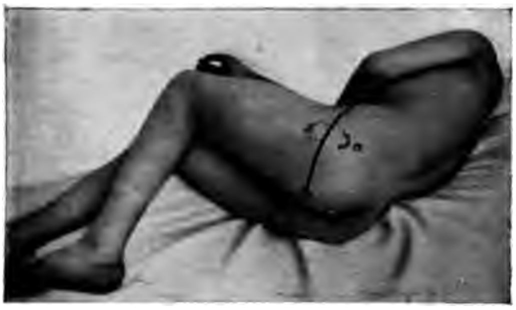


FIG. 443.—Estimation of the level of the trochanter by means of Roser-Nélaton's line in a case of coxa vara. *a* = Actual level of the trochanter (here raised.) *b* = normal level of the trochanter.

The level of the trochanter can be estimated rapidly and reliably in three different ways :—

Roser-Nélaton's line, which joins the tuberosity of the ischium with the anterior superior spine of the ilium, is determined with the hip semi-flexed (135). The trochanter lies normally in this line. If it is above this the level of the trochanter is said to be raised (fig. 443). A displacement of ½ to 1 cm. is not considered pathological, as it comes within the limits of error of measurement.

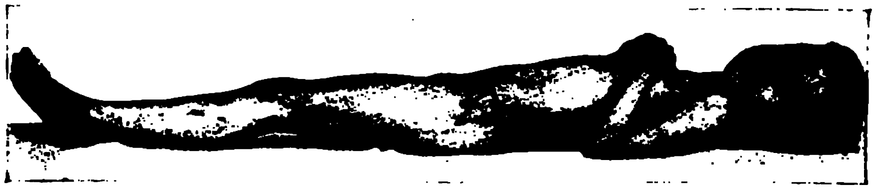


FIG. 444.—Measurement of Bryant's triangle in case of unilateral congenital dislocation of the hip. Normal side. The triangle is isosceles.

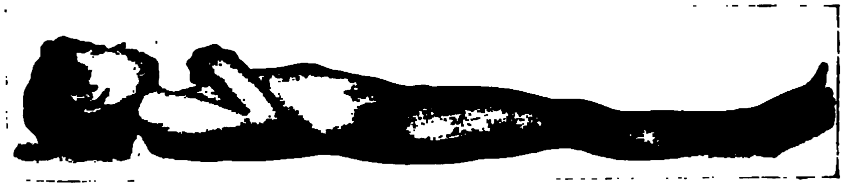


FIG. 445.—Same case. Dislocated side. The triangle is acute angled. The comparative measurement of the horizontal base gives the amount of displacement.

Bryant's triangle is also employed, and is constructed in the following manner : The patient lies flat on his back and the axis of the femur is prolonged with a blue pencil line above the trochanter. A vertical line is then drawn from the anterior superior spine to this base line, and another line is drawn joining the iliac spine with the tip of the trochanter. The right-angled triangle so obtained is normally an isosceles triangle, whereas the base line formed by the prolongation of the axis of the femur is shortened compared to the opposite side, when the level of the trochanter is raised (figs. 444 and 445).

Shoemaker's method is even more simple, and it consists *of prolonging the line joining the trochanter with anterior superior spine, on to the abdomen.* This prolonged line normally meets the median line at the level of the umbilicus or above it; but when the level of the trochanter is raised it intersects this line below the umbilicus (fig. 446).

The head of the femur does not leave the capsule in cases of congenital dislocation as it does in traumatic displacement. The capsule becomes dragged out, in the shape of a pocket, and is displaced posteriorly with the head. The thigh does not, therefore, assume the typical, mathematically fixed position in relation to the pelvis which it presents in cases of traumatic dislocation. Indeed the drawn-out capsule permits abnormally free play for the movements of femur. Congenital dislocation of the hip is therefore characterized by remarkably great *acrobatic mobility of the thigh,* and there is no pathognomonic position in cases of congenital dislocation of the hip in young children. Freedom of movement only diminishes after the lapse of years, but even then, abnormality of position is much less pronounced than in cases of traumatic dislocation.

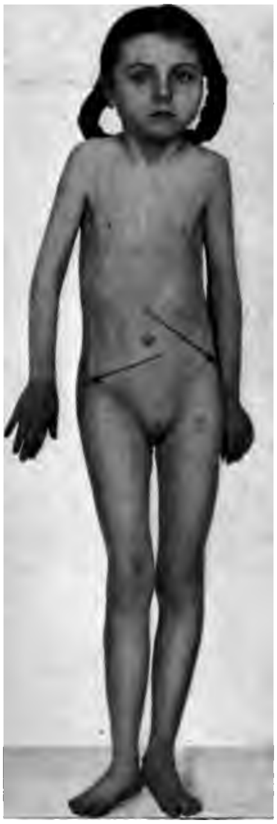


FIG. 446.—Same case. Determination of level of trochanter by prolonging towards the umbilicus the line which joins the trochanter to the anterior superior spine.

Another important sign, connected with this defective fixation of the head, is the *possibility of moving it backwards and forwards on the* *pelvis.* This sign is never absent in cases of congenital dislocation of the hip, if they are not too old, but it can only be demonstrated in refractory children when the muscles are relaxed by anæsthesia. Before proceeding to this test, we must endeavour to show that the *head is in an abnormal position*, and we shall generally find it above or behind the acetabulum.

In the case of somewhat elderly thin girls it is quite possible to see the head of the bone moving about, with every step, in the gluteal region. But it is not evident externally in very small fat children, nor can it be readily felt, especially if it is not fully developed. Sometimes anæsthesia is indispensable for the examination, but whether employed or not, we proceed in the following manner, as taught by Malgaigne (fig. 447). The child is laid upon the healthy side; the leg of the deformed side is flexed to a right angle, and, if necessary, is somewhat adducted. Attempts are made by pressure on the femur from the knee, with one hand, to press the head of the bone as far as possible away from the pelvis. At the same time movements of rotation are made with the same hand, while the upper

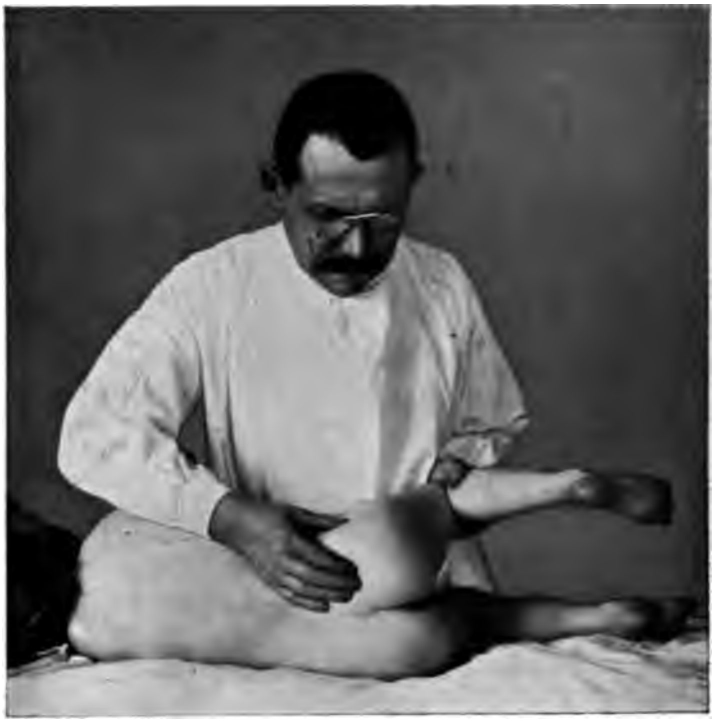


FIG. 447.—Palpation of the head of the bone in congenital dislocation of the hip.

end of the femur is being palpated by the other hand. If only *one* protuberance is felt, this is the trochanter, and there is no dislocation present. But if *two* protuberances are felt, one must be trochanter, and the other is the head of the bone, and a dislocation does exist.

Children who have already walked for some time present another important symptom, especially when the dislocation is bilateral, i.e., a great degree of *lumbar lordosis*, which depends upon the rotation of the pelvis forwards, upon its transverse axis.

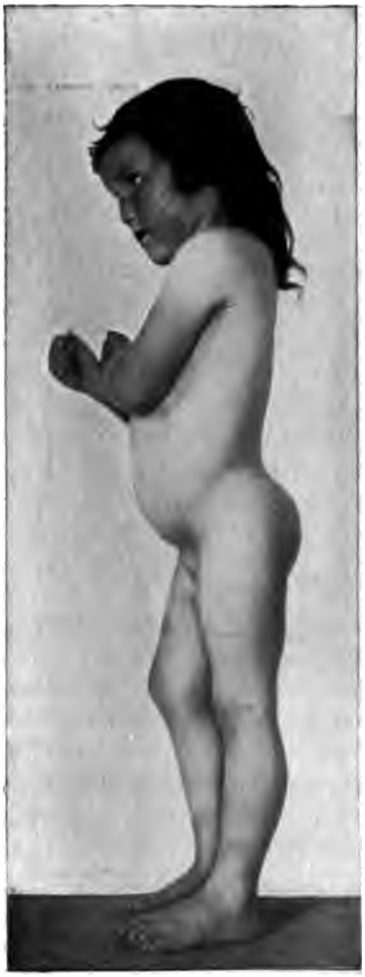


FIG. 448.—Congenital dislocation of the hip. (Lordosis !)

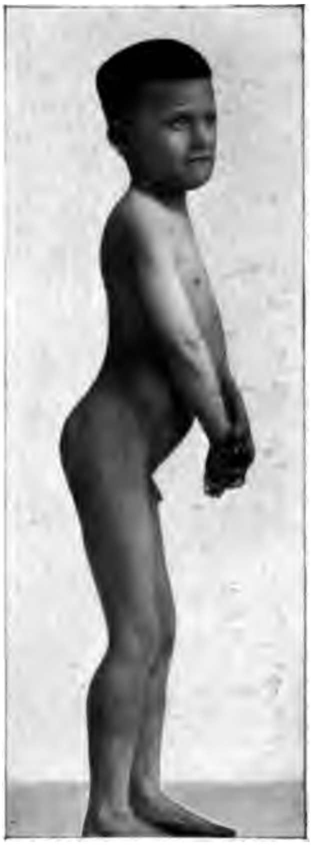


FIG. 449.—Loidosis in progressive muscular atrophy.

The dislocation causes the centre of gravity of the back to fall along a line anterior to the points of body support, and the endeavour to bring it back again over the feet is the cause of the lordosis.

The diagnosis of congenital dislocation of the hip is established if there is shortening, with abnormal mobility of the thigh, elevated level of the trochanter and displaceability of the femur on the pelvis, and if a protuberance is felt besides the trochanter, and if, in addition to all this lumbar lordosis is also present.

A skiagram can only add a few details to the diagnosis, in regard to the shape of the acetabulum and the head of the bone.

We have not yet referred to the *history*, which usually relates that nothing was noticed in the first year, but that a slight lameness was observed as soon as the child attempted to walk. If the dislocation is bilateral, the gait will be described as waddling, and it will have been attributed to weakness; but the disability will be said to be constantly increasing.

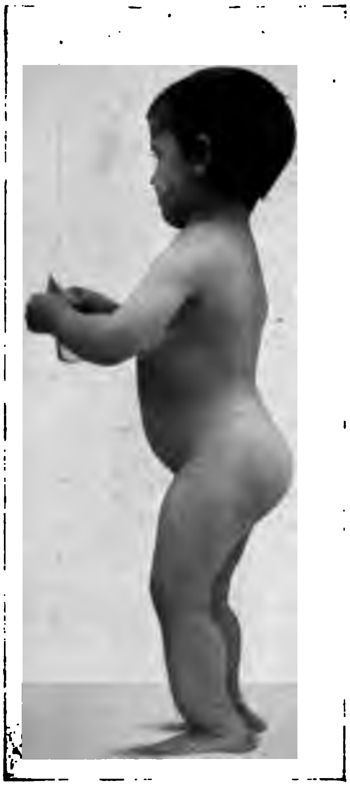


FIG. 450.—Rachitic coxa vara, with simultaneous bending of the shaft of femur and tibia outwards and forwards, and compensatory lordosis.

As we have already seen, the signs are so striking, and can usually be demonstrated with so much accuracy that it is hardly possible to overlook a definite case, and the favourite diagnosis of a past generation—"weakness of the muscles and bones"—ought never to be made by a modern practitioner. An error in regard to a slight degree of *bilateral dislocation* is perhaps excusable. In such a case, we are unable to judge of abnormal mobility and shortening by comparison with the healthy side. We are only able to diagnose the dislocation, apart from the lordosis and the duck's gait, by the movability of the femur on the pelvis, the elevated level of the trochanter, and the detection of the head of the bone, above or behind the acetabulum. Both types of cases sometimes require examination under an anæsthetic. The only affection with which confusion is conceivable is a rachitic curvature of the femur, especially if this is accompanied by a bilateral *coxa vara*. Such children often present a pronounced lordosis, i.e., they protrude their abdomen forwards, in order to regain the equilibrium, which is disturbed by the curvature of the femur (fig. 450). The gait is often awkward, and resembles the waddling in the case of congenital dislocation of the hip. The similarity of the conditions is enhanced by the fact that the level of the trochanters is raised in coxa vara and because they project abnormally far at the sides (see shape of hips in fig. 452).

But an important difference is afforded by the abnormal mobility in congenital dislocations, in contrast to the restricted movements in coxa vara. There are, however, cases of coxa vara wherein the restriction of movements is but slight, namely, the coxa vara of little rachitic children, and, on the other hand, there are cases of congenital dislocation—mostly in older children—wherein the abnormal mobility has become diminished. If, finally, the head of the femur is only slightly developed, and is therefore difficult to feel as a separate structure, and if the dislocation is not definitely of the iliac variety, but of the supracotyloid variety, which, exceptionally, may remain in that position until an advanced age, it is quite conceivable that the case may be obscure until an examination is made under an anæsthetic. Trendelenburg has indicated a sign which may be valuable in such a case. He points out that when a normal individual stands on one foot, the opposite half of the pelvis is raised, on account of the muscular fixation of the joint. In an individual with congenital dislocation, this fixation of the joint is absent, and therefore the opposite half of the pelvis sinks. However, if there be a dislocation, anæsthesia will enable the head to be felt, and the oscillating move-

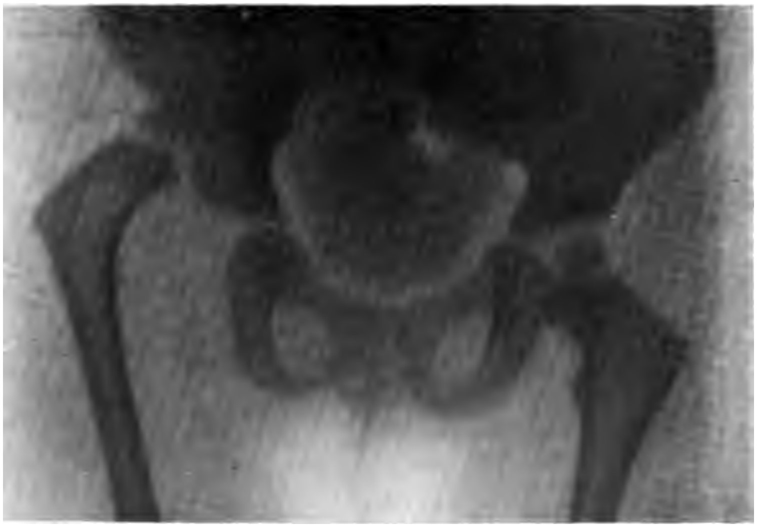


FIG. 451.—Congenital dislocation of hip.

ment to be detected. If doubt still remains, it can be finally cleared up by X-ray examination.

Infantile paralysis, which has affected the gluteal muscles, may involve the beginner in difficulty. The lameness is very similar to that of dislocation (paralytic limp). The trochanter becomes so prominent, owing to the atrophy of the gluteal muscles, that the inexperienced is apt to mistake it for the head of the femur. Owing to the muscular paralysis the passive movements of the limb are very extensive, and in old cases there may be some shortening. But the history in infantile paralysis is that the little patient previously walked quite well, that he became ill with infective symptoms, and had, at first, widespread paralysis which gradually subsided somewhat.

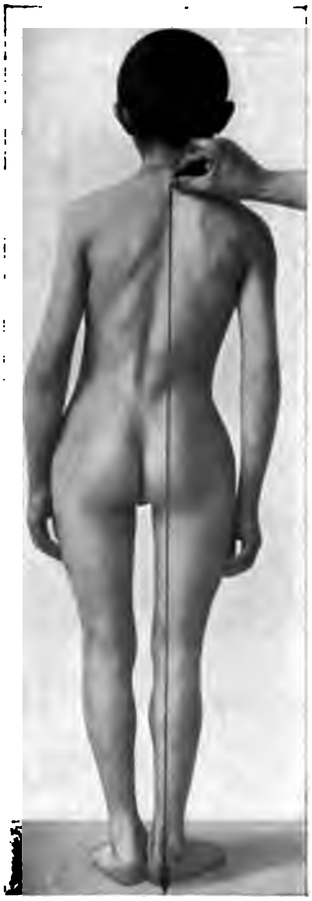


FIG. 452.—Bilateral rachitic coxa vara. Extreme projection of the trochanter region.

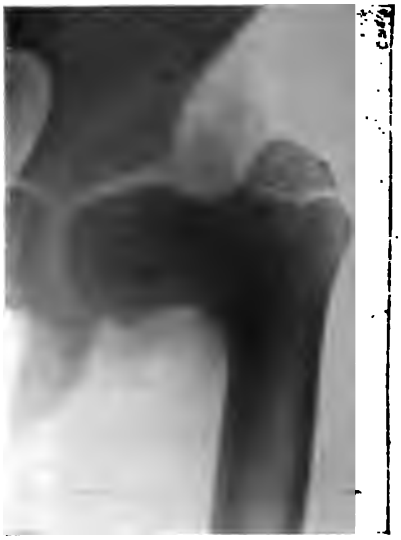


FIG. 453.—Rachitic coxa vara. (Skiagram of fig. 452.)

Physical examination at once shows that the femur is not displaced on the pelvis, and that the prominence seen under the glutei is not the head of the femur, but an abnormally evident trochanter. Although the limb may be shortened, the level of the trochanter is not raised, showing that the whole femur is shortened and not merely displaced upwards, as in dislocation.

Sometimes genuine dislocations are associated with paralysis

—not congenital, but acquired *paralysis*. In infantile paralysis they are unilateral, and depend upon the traction of the unparalysed, but un-antagonized adductors. In congenital spastic paraplegia—Little's disease—the dislocations may be bilateral, due to spastic contraction of the adductors. The paralysis may dominate the clinical picture so that the dislocation may be entirely overlooked, especially in bed-ridden children, although, owing to the muscular atrophy, this could easily be detected on grasping the hip.

Progressive muscular atrophy (fig. 449) is a disease which at first sight may suggest congenital dislocation of the hip because of the lordosis. A careful examination will, however, quickly expose the error.

Congenitally dislocated hips are frequently affected in the course of time with inflammatory processes of the nature of *arthritis deformans*, causing considerable pain. This may impel the patients to seek advice, rather more than for the deformity—a circumstance which may be of diagnostic significance.

A female patient, aged 24, sought advice for "rheumatic" pains in the right hip. She was of normal girth, but short in stature, and she had a strikingly stiff, peculiar measured gait. She had suffered with her hips from infancy, and had always been treated for "general weakness." Examination showed a congenital bilateral dislocation of the hip, with arthritic changes on the right side. The skiagram showed that the heads of the femora were displaced to the extent of 9 cm., and that a new joint had developed on either side of the pelvis.

The slow stiff gait was very remarkable in this case. Her æsthetic taste had unconsciously and gradually adapted it to such an extent as to completely conceal the waddling. Some female patients entirely lack this compensatory influence, and the term "duck's gait" hardly does justice to what is seen in some advanced cases.

The following case is equally significant. A female patient, aged 36, in whom some "weakness" of the left leg had already been detected in childhood, began to suffer pain in the left hip and to limp when she was 31. She went from one practitioner to the other, and was considered to be a case of "rheumatism." This diagnosis did not, however, explain the shortening of 1½ cm. Palpation did not reveal anything definite, because of the patient's stoutness. A skiagram had to be made in order to distinguish between the results of an old hip disease, or of an injury, and congenital dislocation. The skiagram was in favour of the latter. It was not the dislocation which caused the patient to seek advice, but the secondary arthritis.

B.—COXA VARA.

If the head of the femur is not in an abnormal position, and if the bone cannot be displaced on the pelvis, there cannot be any dis-

[illegible]

[illegible]

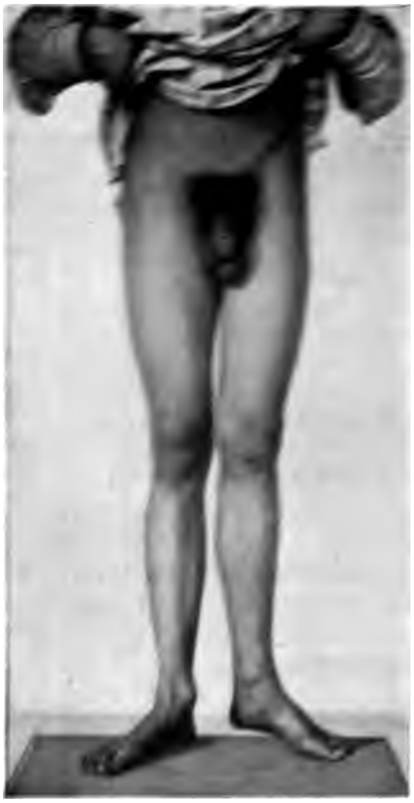


Fig. 434. Left-sided coxa vara in a youth.

[illegible] very rare [illegible] congenital coxa

[illegible]

In cases of rickets, the [illegible] usually bilateral, but [illegible] has been caused [illegible] abnormal weight-bearing [illegible] coxa vara of adolescents, it may be either unilateral or bilateral.

But even in this case we must assume some abnormality in the constitution of the bone. It matters very little whether we classify it with late rickets, or designate it in some other way, because we are not fully acquainted with the nature of late rickets. But in any case there is no justification for classifying all cases of coxa vara of adolescents as traumatic, as is sometimes done. This would mean that any weight acting upon the neck of the femur could be regarded as a trauma.

Let us first consider in detail a case of *rachitic coxa vara* in a child. The posture of the leg and foot is as normal as the rachitic curvature permits, but we are struck by the extreme projection and the great elevation of the trochanters. The gait of the little patient is exceedingly clumsy, and may sometimes be characterized as slightly waddling. The movement of the projecting trochanter, which is visible under the skin, resembles somewhat the movement of the head of the femur under the gluteal muscles in congenital dislocation of the hip. But, as already stated, the resemblance is merely superficial, for in coxa vara there is only *one* protruberance, whereas in dislocation there are *two*. On examining the individual movements we find that there is freedom of flexion, that external rotation is normal or only slightly restricted, but that abduction is diminished or almost impossible. The only abnormality shown by the skiagram (fig. 453) is adduction of the neck of the femur towards the shaft, usually to an angle of about 90°—*coxa adducta*.

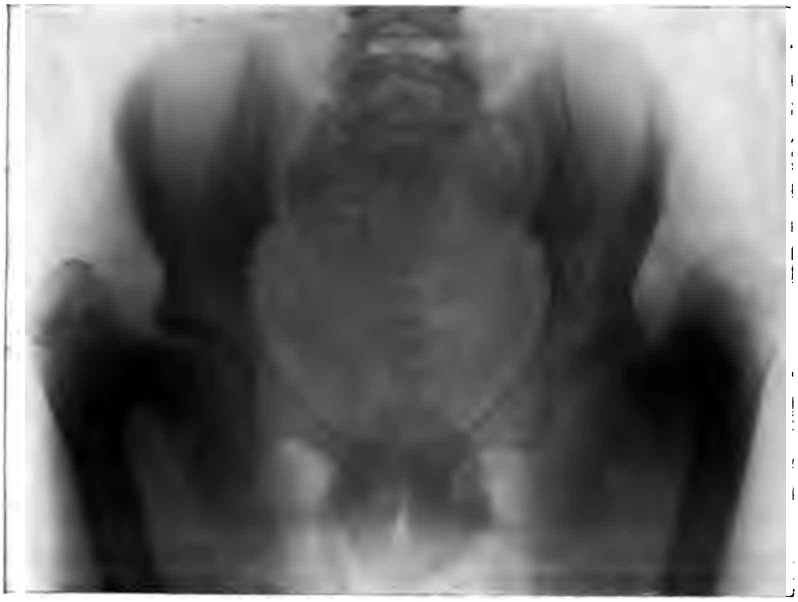


FIG. 455.—Severe bilateral coxa vara, patient, aged 15. Rachitic form.

If we examine the rest of the skeleton we are almost sure to find other signs of rickets in the bones (fig. 450).

The restriction of abduction varies. In young children it is slight, but later on, between the eighth and tenth year, it may be very pronounced and constitute the most troublesome symptom.

In unilateral coxa vara the distance between the anterior superior spine and the malleolar is shortened in the affected limb, and there is corresponding *unilateral lameness*. This is, however, a condition which is much rarer in rickets than the coxa adducta which occurs after fractures of the neck of the femur, osteomyelitis, or tubercle.

A more complicated clinical picture presents itself in children, in cases of *coxa vara in the strict sense of the word,* i.e., bending of the head downwards and backwards, often with simultaneous twisting of the neck.

For the head to retain its normal relation to the acetabulum, the shaft of the bone, and therewith the whole limb, would have to assume a position of adduction, external rotation and hyperextension. In order to improve upon this impossible position, the limb—head and shaft—executes a movement which is composed of abduction, inward rotation and flexion. The more pronounced the deformity, the more is the whole range of the movement in these directions taken up in merely attaining a normal posture, and the less is it possible to abduct, invert or flex the limb any further. These simple considerations explain the disturbances of function, which we meet in this form of coxa vara.

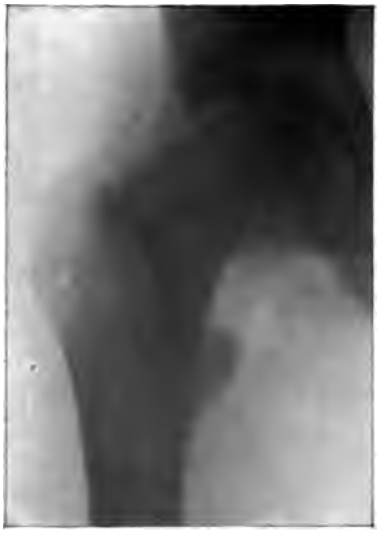


FIG. 456a.—Coxa vara in a youth. Bending in the vicinity of the epiphyseal line.

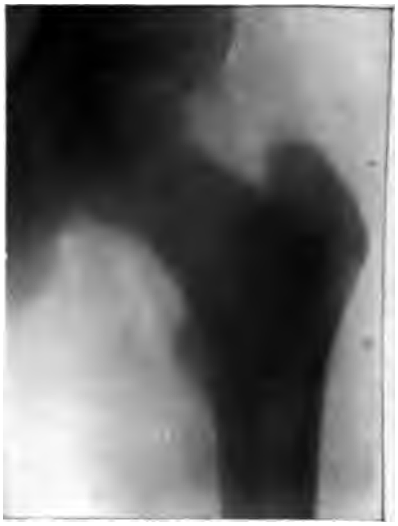


FIG. 456b.—Healthy side in same patient.

The gait of a patient with a unilateral affection presents the same lameness as a case of painless stiffening and shortening of the limb. The pelvis is pushed forwards at each step and the limb proceeds as a rigid whole with the pelvis. When the affection is bilateral, there is the peculiar gait wherein the pelvis oscillates around a vertical axis with every step. We will also notice that the affected limb is always held in a position of eversion, an obvious result of the downward inclination of the head, which is no longer fully compensated for. The skiagram shows mainly a downward displacement of the head, in the epiphyseal line, on a normally placed neck (fig. 456).

We may summarize what has already been said, in regard to *coxa adducta*, as follows: *Prominence and elevation of the level of the trochanter, restricted abduction, and shortening, if the affection is unilateral.* In regard to *coxa vara in the strict sense of the term*, we may summarize thus: *Prominence and elevation of the level of the trochanter with external rotation and restriction of abduction, internal rotation and flexion; and shortening with lameness of one side if the affection is unilateral.*

Sometimes unilateral coxa vara in a youth is mistaken for *commencing hip disease.* Coxa vara has, like flat foot, a painful stage which may easily be regarded as some inflammatory affection.

A young man (fig. 454) sought advice for commencing "hip disease." He limped slightly towards the left, but two facts were noticeable at first sight: the limp was not painful, as he planted his left foot down quite firmly without any special care. It rather appeared to be due to shortening. In addition, the foot was considerably everted. This, of course, excluded hip disease. The stage wherein eversion exists is accompanied by some abduction and apparent lengthening. This stage is also so painful that it is impossible to bring the foot down to the ground, without special care. Our thoughts, therefore, proceeded in another direction. We measured the leg and found 2 cm. of shortening, which is hardly possible in early hip disease. The level of the trochanter was definitely raised. On testing the movement it was found that abduction, flexion and inward rotation were restricted. This established the diagnosis of coxa vara, and the skiagram could do nothing but confirm it.

Complete rest with the application of an extension apparatus relieve the painful symptoms much more quickly than in the case of hip disease, which affords a further proof of the accuracy of the diagnosis.

CHAPTER XCV.

ACUTE INFLAMMATORY DISEASES OF THE HIP-JOINT.

A PATIENT is suddenly seized with severe pains in the hip, and is unable to move his leg with freedom. We have excluded acute disease of neighbouring structures, such as phlegmonous inflammation of the crural or inguinal glands, or an acute abscess of the pelvic fossa. It therefore only remains to decide whether the case is one of acute arthritis of the hip-joint, or osteomyelitis of the shaft of the femur, or even of the pelvis.

It is true that there is pain with every movement in *osteomyelitis*, just as there is in hip disease, but nevertheless passive movements may be carried out within certain limits, if due care is exercised. The local pain on pressure does not correspond to the region of the joint, but has its highest point at some distance from it below the trochanter, at the extreme limit of the osteomyelitis—or, on the other hand, upwards towards the pelvis. If there is secondary involvement of the joint the symptoms of the two diseases coalesce, but we ought to be able to decide the sequence of events by the fact that the pain on pressure extends comparatively far either upwards or downwards, and by the thickening of the bone. In acute hip disease, however, including a circumscribed osteomyelitis of the head and neck of the femur, we are struck from the very beginning with the intense painfulness of any passive movement of the joint, if not by its complete muscular fixation, no matter in which position this fixation has taken place. The maximum pain on pressure is in the vicinity of the head of the bone, i.e., beneath the middle of Poupart's ligament. On inspection and palpation it may be noted that this region is fuller than on the healthy side. The pyrexia proves that some acute inflammatory disease exists.

What is the nature of this inflammation? If several other joints are involved simultaneously, or in rapid succession, we should think of acute articular rheumatism—but one must be sure that only the joints are affected. If the disease is limited to the hip, and the patient is young, the condition is most probably one of acute osteomyelitis of the neck and head of the femur, involving also the joint. This diagnosis becomes quite certain if other areas of bone disease exist, or if some suppurative inflammation is actually present at the time, or has recently subsided.

If the patient is a child convalescent from scarlet fever, we know that the case is one of scarlatinal arthritis, which may recover without incision, and with complete restoration of function, or which may rapidly destroy the joint. Similar forms of inflammation of the hip may more rarely come on, after typhoid fever, small-pox or measles. The hip is sometimes involved in puerperal articular disease. In default of any of these more frequent causes, we must seek other possible portals of entry for organisms of inflammation, and we should not forget gonorrhœa. Subacute inflammation of the hip may occur in secondary syphilis and in the congenital disease, but it is rare. Sometimes it is quite impossible to determine how the infection entered, or to discover any cause.

The organisms of acute suppuration sometimes give rise to a form of inflammation which is mistaken for tubercle, because staphylococcic and streptococcic infections of the neck of the femur do not always produce their familiar clinical picture. On the contrary, the disease may run such a mild course, despite its acute onset, that it easily suggests tubercle, unless a very reliable history is at hand. The records of von Brun's clinic have shown that this mistake is by no means rare.

The following is a typical case :—

We were consulted about a lad aged 12, who presented all the symptoms of early hip disease—moderate pain on axial pressure, and on pressure over the trochanter, fixation of joint in a position of slight flexion, adduction and inversion ; temperature practically normal. This condition had existed unchanged for several weeks. Had we had no reliable history, we might have assumed that the case was tubercular. But as a matter of fact, the patient had been admitted to hospital with the diagnosis of acute osteomyelitis of the upper end of the femur. The disease had started suddenly, with severe, though transitory, fever. We accepted the diagnosis of the family practitioner, and it was confirmed during the course of the next month by the development of osteomyelitis of the shaft of left humerus, with sequestrum formation. The lad had complained of pain in the left arm from the beginning, but he laid no stress on it, as his hip symptoms were much more distressing.

One word in reference to the **results** of these inflammations. As long as the measurement between the anterior superior spine and malleolus is of normal length, having regard to the position of the leg, and the tip of the trochanter is at the correct level, the case is one of pure *arthritis.* But if there is sudden shortening, or if the trochanter becomes displaced upwards, there must be some *pathological dislocation,* usually backwards, or a *separation of the epiphysis.* The former is more frequent after typhoid or scarlet fever ; the latter, after osteomyelitis, in which condition a fracture near the shaft may occur. The actual state of affairs can be determined by careful examination.

If the limb is slightly everted and can be moved hither and thither on the pelvis, and if soft crepitus can be felt, a separation of the epiphysis is present. If the leg is in the position of a posterior dislocation, i.e., flexion, adduction and inversion, and if the head of the femur is felt under the gluteal muscles it is obvious that a dislocation is present.

There is yet a further possibility, which is, however, rare in acute arthritis, but may lead to an error of diagnosis.

A little girl was suffering from scarlatinal hip disease and her limb was shortened and in a position of eversion. It was possible to move the femur on the pelvis, and soft crepitus was felt. We diagnosed inflammatory separation of the epiphysis. The skiagram, however, showed severe destruction of the upper border of the acetabulum and partial destruction of the head of the femur, which was not detached. The case was really one of pathological dislocation in which there was no pathognomonic malposition, and in which there was no possibility of successful reduction, in consequence of the destruction of the joint. As soon as the residue of the head was brought into a normal position, it glided backwards again, because of the destruction of the upper border of the acetabulum. An operation was performed subsequently and this confirmed the X-ray diagnosis.

CHAPTER XCVI.

CHRONIC INFLAMMATORY DISEASES OF THE HIP.

Chronic inflammatory diseases of the hip are mainly comprised within two groups. The one includes tubercular coxitis, the other embraces all that is understood by the term chronic rheumatic arthritis. There are other rare conditions, which will be referred to when discussing differential diagnosis. An approximate distinction is furnished by the circumstance that the tubercular disease affects growing individuals, while the non-tubercular arthritis affects adults.

A.—TUBERCULAR HIP DISEASE.

The diagnosis of tubercle is forced upon us if a child begins to complain of feeling tired and has a painful limp after walking for some distance, if the gluteal fold is obliterated and some muscular atrophy on the affected side is also possibly noticeable. As the limp is not permanent at first, but only comes on when the patient is tired, and as it can be suppressed when an effort is made to do so, it has been called a "voluntary limp"—an expression which constitutes an unjustifiable reproach. The progress of the case is a comment on its "voluntary" nature. Sometimes the child complains more about the knee than about the hip, but a cursory examination will show that the knee-joint is free.

Having examined the gait of the child with his clothes off, we lay him upon a flat table and direct him to extend both legs completely. A hollow is observed to form in the back, so that the hand can be passed flat under the lumbar region. If we make the back fit closely against the table, the knee of the affected side becomes slightly raised. This preliminary examination suffices to show that the hip is held in a constrained position of slight flexion, although otherwise the limb may either be abducted and everted, or adducted and inverted, both positions being met with in the early stages.

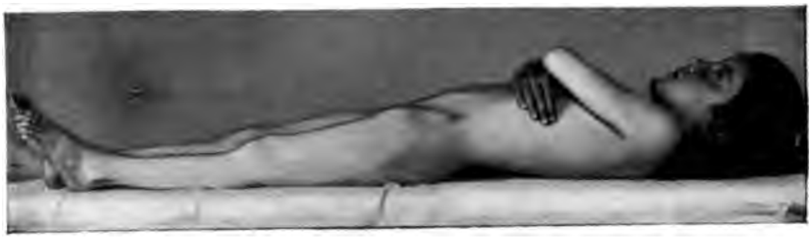


FIG. 457.—Left-sided hip disease. The position of slight flexion compensated for by the lumbar lordosis.

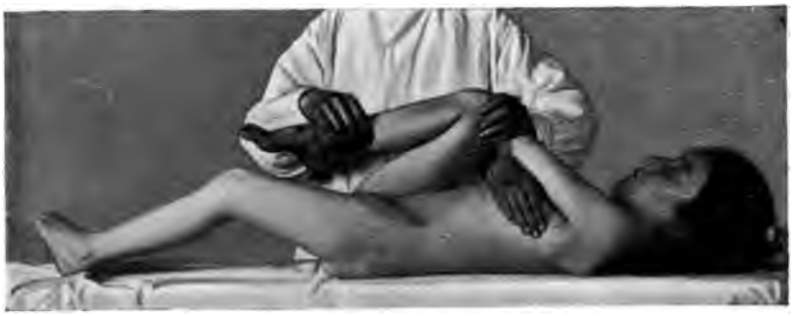


FIG. 458.—Same case. The flexion demonstrated by Thomas's manœuvre.

If we cannot get the back to lie close against the table, we may adopt the simple and painless method of Thomas, i.e., we flex the *healthy* hip as much as possible in order to throw the pelvis backwards, and thus the spinal column will certainly be forced close against the table. If there is the slightest degree of flexion on the diseased side, the knee will then be raised, so that, at any rate, the hand can be passed under it (figs. 457 and 458).

We then proceed to a careful examination of the separate movements, as compared with the healthy side. The more limited flexion, extension, adduction, abduction, external and internal rotation are, the more does the patient endeavour to transfer these movements to the lumbar spine, and he moves his pelvis and leg as one fixed whole. In other words, the pelvis participates in the movements. We must then inquire whether this fixation depends upon pure muscular spasm, or upon any organic changes in the joint. If the degree of fixation after a long rest differs from the degree present when the patient is very tired, it would indicate a muscular origin, but if there is no difference, the rigidity must depend upon causes within the joint. An absolute decision can only be arrived at by examination under an anæsthetic

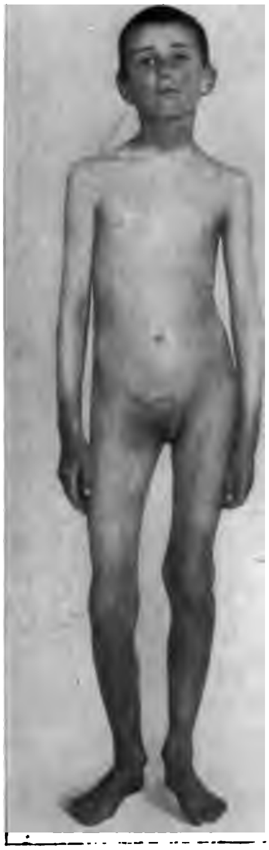


FIG. 459.—Early stage of hip disease on right side, *with external rotation, flexion, and abduction.*

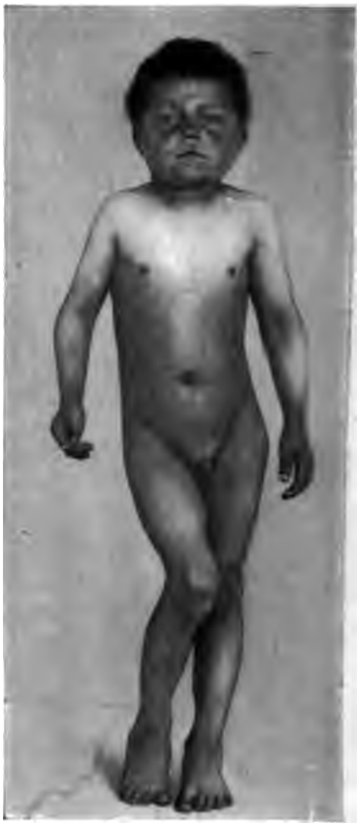


FIG. 460. Early stage of hip disease on right side, *with flexion, adduction, and inward rotation.*

when muscular fixation vanishes forthwith without any forcible manipulation. Abduction and rotation are the movements which are generally first interfered with. If the adductors become tense on attempting to perform abduction rapidly, the hip-joint is certainly involved, even if extension and flexion remain perfectly free.

It may happen in this stage that all symptoms disappear after a few weeks' rest in bed, so that one thinks an error in diagnosis has been made. But the symptoms return in a few months, or even after a few years, and the classical picture of hip disease develops.

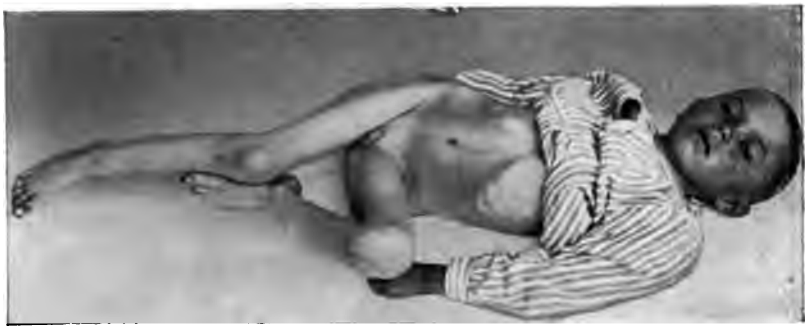


FIG. 461.—Advanced hip disease of left side with extreme flexion and abduction.

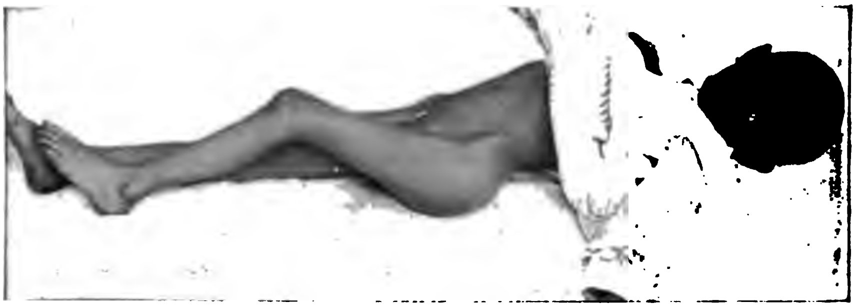


FIG. 462.—Advanced hip disease of left side, with internal rotation, adduction and flexion.

If we are not quite clear about the limitation of abduction we direct the patient, after taking off his clothes, to spread out his legs as much as possible while standing. Any asymmetry which then exists in the posture of the leg or back is an indication that abduction is interfered with.

A certain amount of importance attaches to the division of hip disease into *stages, in accordance with the posture* of the limb. As König has shown, the patient endeavours to spare his hip-joint as much as possible in all stages of the disease. For this purpose the position of slight abduction and external rotation, with a little flexion, is the most useful, as long as the patient walks without crutches (fig. 459). But if the patient does go about on crutches, he raises his diseased leg, that is to say, he flexes it more, but still holds it in abduction. If the patient takes to his bed in the earlier stage of his disease, he supports his flexed, diseased leg on the healthy one, thus bringing it into the position of adduction and internal rotation (figs. 461 and 462).

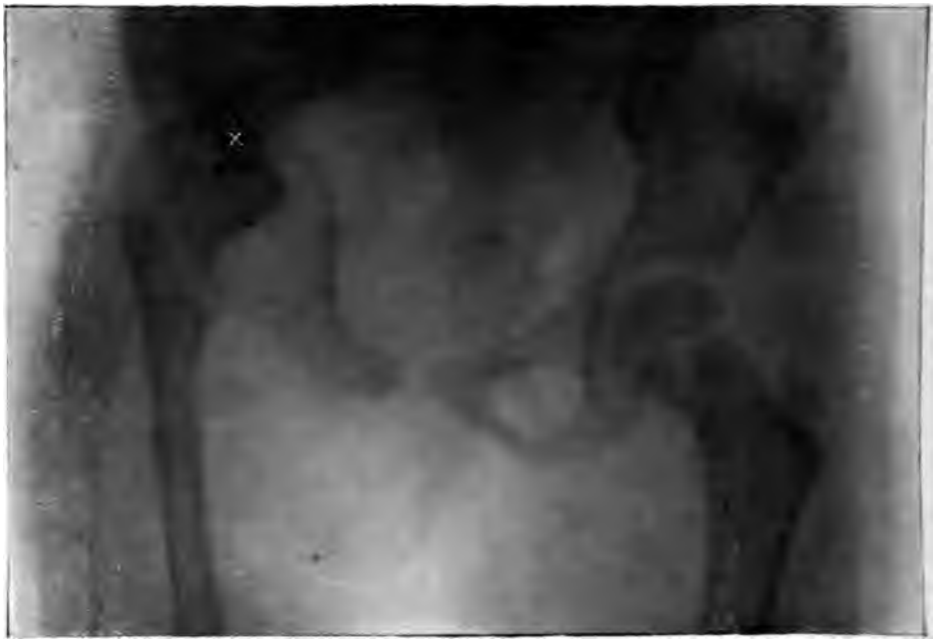


FIG. 463.—Hip disease with "displacement of acetabulum" and coxalgic pelvis. (Skiagram of fig. 457.) Small sequestrum at X.

There is an *anatomical* cause for this position, if the head of the bone has left its normal situation and has become displaced posteriorly, or backwards and upwards, either through destruction of the capsule or gradual destruction of the posterior border of the acetabulum (so-called displacement of the acetabulum [Pfannenwanderung] fig. 463). The position then assumed is one of subluxation or even of dislocation.

After testing the power of movement we proceed to *palpation*, which, combined with inspection, will show whether any abnormal swelling exists. The principal swellings to be thought of are enlargement of the inguinal glands and abscesses which have made their way to the surface. These usually appear in front (see fig. 457, where there is an abscess below the anterior superior spine), but also occur on the outer and on the posterior surface of the hip.

We next examine for *pain*.

We test for pain on direct pressure, where the joint is most accessible, i.e., in the front, just below the middle of Poupart's ligament. This pain is often an early sign of hip disease, although it is not so significant as loss of power of movement. Importance also attaches to indirect pain, elicited by force applied in the long axis of the femur or to the trochanter. We have already seen that any movement, carried out to an extreme degree, is painful.

The differential diagnosis raises the following considerations :—

(*a*) If it is quite evident that there is *actual disease of the hip-joint itself*, we must first exclude the *subacute forms* of *infective hip disease*. For this purpose it is necessary to give due weight to all the points advanced in the previous chapter, and only diagnose tubercle in the absence of any other cause.

As already remarked in the previous chapter, an acute onset of hip symptoms indicates an acute infective origin, even if the disease, develops into a chronic condition. The sudden rupture of a tuberculous periarticular focus into a joint also causes acute symptoms, including fever, but as a rule this event would have been preceded by slight articular symptoms, pointing to tubercle. A rigor, or herpes labialis, accompanying the acute exacerbation, are indications *against* tubercle.

If the onset is very gradual, the possibility of tubercle must be entertained even if the hip symptoms have followed some acute infectious disease. A child who begins to limp in the slightest degree a few weeks after measles is probably the subject of early hip disease, the measles having prepared the soil for the tubercle.

We must next exclude the so-called *chronic rheumatic arthritis*, which we shall deal with at the end of the chapter.

We have already seen that *congenital dislocations of the hip* and that *coxa vara* are subject to irritative stages, wherein some confusion with hip disease is quite conceivable. The same applies to the hip symptoms, which eventually arise in fracture of the neck of the femur, especially in children.

We should think of primary or secondary *malignant growth*, including hydatid cyst, if the features of the case do not completely accord with hip disease, especially if the slight limitation of movement and its relative painlessness present a striking contrast to the severe and spontaneous radiating pains.

(*b*) *Diseases, independent of the joint*, may simulate hip disease, by causing flexion of the hip and pain in its vicinity, leading to a painful limp.

The most frequent cause of spastic flexion of the hip, not due to joint disease, is a *burrowing abscess from a tubercular spine*, or *pelvic tubercle*. This condition may resemble hip disease so closely that examination of the spinal column and palpation of the pelvic fossa are always essential in cases of spastic flexion of the hip.

The diagnosis is easy if a dorsal curvature is evident, or if an abscess is found filling up the iliac fossa. But matters are not always so simple. The curvature may be quite absent in adults, and the connection of the abscess with the spinal column may be limited to a narrow sinuous track. In abscess due to spinal disease, however, abduction and rotation are usually free at the hip, whereas these movements suffer first if the joint itself is affected. A conclusion may also be based on a skiagram.

In rare cases, a *paranephritic* or *appendicular abscess* may cause spastic flexion of the hip, and an inadequate examination may lead to a wrong diagnosis.

Effusion into the iliacus bursa may also lead to a wrong diagnosis because of the spastic flexion and the swelling immediately over the hip-joint. But as abduction, adduction and rotation are quite free, despite the spastic flexion, such an error ought not to be made.

Unless attention is paid to the free mobility of the joint, *sciatica* and *periarticular neuralgia* are liable to be mistaken for early hip disease, more especially in connection with gynæcological conditions. In some cases of *hysteria* there appears to be a real contracture at the joint; but symptoms usually vanish on correct treatment by suggestion.

It is sometimes difficult, in the case of a young girl, to decide at the first examination whether the condition is hysterical or the early stage of hip disease. The family history as well as the previous history must be taken into consideration. I have seen a girl simulate several tubercular joints in the course of a year, but the contractures rapidly disappeared after appropriate psychical treatment. In another case, I was at first inclined to diagnose hysteria, because the symptoms disappeared at times ; but the sequel showed that hip disease really existed.

The foregoing considerations having led us to the diagnosis of tubercular hip disease, we must next endeavour to determine the *variety* and *degree* of the disease.

The clinical signs do not, as a rule, permit us to say whether the disease has started in the bone or in the synovial membrane. If the course of the disease is intermittent, it suggests the presence of diseased bone close to the joint. If abscesses occur, we must assume that the bone is involved, either primarily or secondarily.

An *X-ray examination* is the best method for the early diagnosis of a diseased area in bone.

If the bony outline is normal, and the gap representing the cartilage is of the ordinary size, but the bone itself is abnormally transparent to the rays (osteoporosis), and the shaft of the femur is narrowed, we may conclude that there is atrophy from disuse, as is usual with tuberculosis. It is, however, impossible to decide whether a primary synovitis exists, or whether a small focus of disease is present in the bone.

If the transparent streaks corresponding to the articular cartilage are narrower than on the healthy side, but the picture is otherwise as just described, we must assume that the cartilage has already become partially absorbed.

If the sharp edge of the head of the bone or of the acetabulum is replaced by a rough irregular border, it means that the cartilage has been destroyed and the adjacent bone eaten away; but even this condition does not exclude a primary synovitis. But whatever the aspect of the outline of the bone may be, there can be no doubt about the existence of a primary focus of bone disease if there is a transparent area, in the head (fig. 464), the neck, or in the pelvis. This area may either be sharply defined or confused in outline, and is sometimes surrounded by a thick zone (osteosclerosis) with a more opaque structure in the centre (sequestrum).

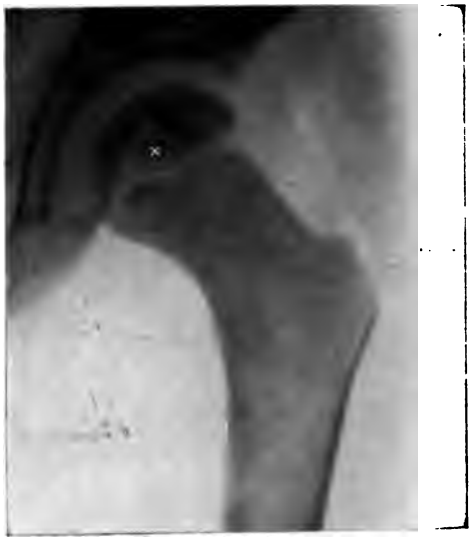


FIG. 464.—Tubercular focus and sequestrum (x) in head of femur.

In the later stages it is necessary to diagnose the *secondary changes*, viz., displacement of the acetabulum, spontaneous dislocation, and separation of the epiphysis. The differentiation of these various processes possesses a certain therapeutic importance.

If the displacement has occurred suddenly, or, at any rate, has been aggravated suddenly, for example, as the result of a slight injury, it indicates either a spontaneous dislocation or a separation of the epiphysis—or even fracture of the neck in the vicinity of a large area of disease. The diagnosis, as between fracture and dislocation, is made on the ordinary principles. It should, however, be noted that when the joint capsule is severely affected the displacement is less and the mobility is greater than in a traumatic dislocation. If the displacement occurs gradually, it means that the capsule has worn away by degrees, and the head of the bone has escaped unnoticed, or that the acetabulum has been gradually changing its position, its upper border being displaced upwards, or upwards and backwards by pressure atrophy (fig. 463). The acetabulum thus loses its circular shape, and assumes that of a fish dish. In either case the limb takes up the position of adduction, flexion, and internal rotation. It is quite impossible clinically to distinguish between a severe degree of displacement of the acetabulum and a dislocation of gradual onset. This is, however, of no importance, because such dislocations, unlike those which occur suddenly, are incapable of reduction.

The distinction may, however, be made by means of a skiagram, which will show at the same time the changes which the entire pelvis has undergone (coxalgic pelvis, fig. 463). The diseased side of the pelvis is inclined considerably forwards in relation to the other side, having rotated on a frontal axis (Hofmeister).

B.—NON-TUBERCULAR CHRONIC HIP DISEASE.

If a patient of advanced years comes complaining of his hip, our thoughts will run in a totally different direction. We must first make sure that the hip is really affected, and that the case is not one of *sciatica*.

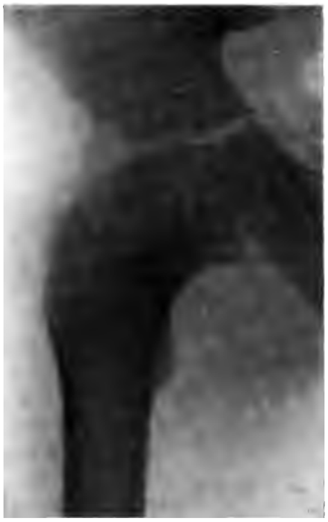


FIG. 465.—Tubercular hip disease. Erosion of head of femur and of acetabulum beginning.

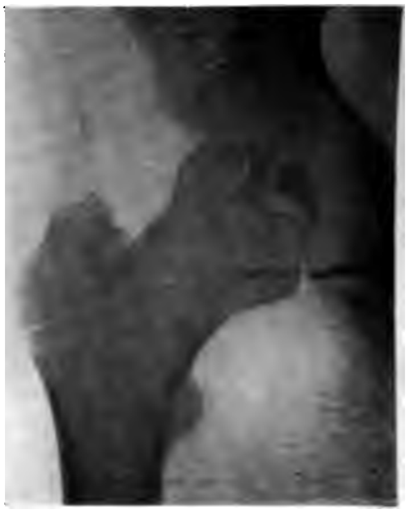


FIG. 466. — Tubercular hip with a wedge-shaped focus of disease in head of bone.

If, despite the long duration of the malady, the limitation of movement and pain are comparatively slight, and there is not much alteration in the posture of the limb, we should think of "rheumatic" arthritis, and should endeavour to ascertain its cause from the clinical history of the patient, as explained in Chapter 83. An examination of the other joints will be useful, but we must not exclude chronic articular rheumatism of the hip, because there is no other joint affected. The so-called "senile hip joint" is usually mono-articular. Clinical examination affords much information about the anatomical condition of the joint, if we estimate carefully the degree of impairment of movement, the measurement of the limb and the presence of grating, &c. An accurate knowledge of the state of the bone can, however, only be derived from a skiagram, which is indispensable in these cases, and which gives the opportunity of correcting a wrong diagnosis.

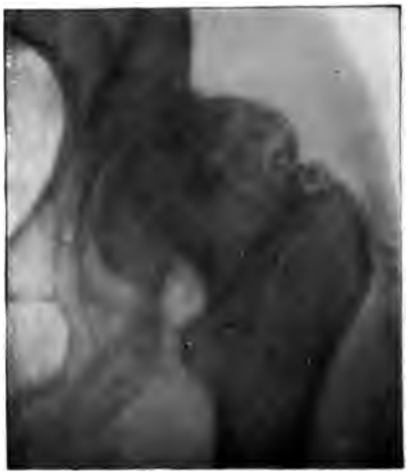


FIG. 467.—Chronic hip disease with deformity. ("Senile hip.")

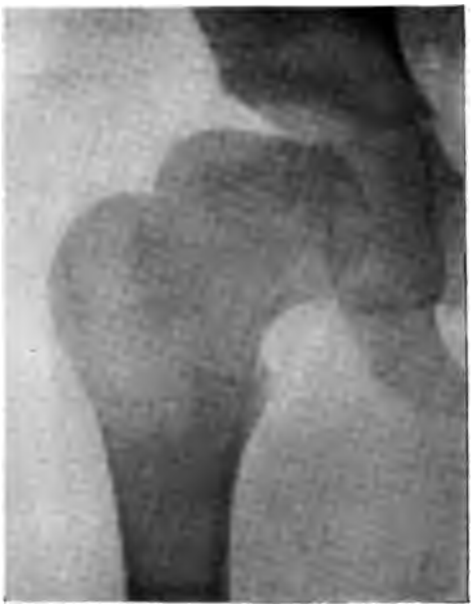


FIG. 468.—Hip disease with deformity in a child.

Anatomically, the most important are those processes which produce *deformity*, partially destructive and partially proliferating in character. The next in frequency are the forms which produce *ankylosis*.

As far as etiology is concerned, the cases wherein the disease remains permanently, or at any rate for a long time localized to one joint, are of more interest to us than those cases wherein the multiplicity of the affected joints facilitate the diagnosis. In the former cases the disease often originates in an injury, sometimes a simple contusion. The younger the patient, the more likely this causation. On the other hand, elderly individuals may suffer from arthritis of the hip with deformity, in the absence of any demonstrable injury, i.e., *senile disease of the hip*.

In the absence of a history of injury, an examination should be made for nervous diseases, especially tabes and syringomyelia.

Although we have regarded adult age as a contra-indication of tubercle, it may still be necessary to fall back upon this diagnosis in cases which are otherwise insufficiently explicable, especially if the patient has a tubercular heredity, or has already suffered from some tubercular condition, and if the pain is very severe, and the disease progresses somewhat rapidly without any temporary improvement. Tubercular hip disease has exceptionally been met with, even at the age of 80.

The diagnosis is sometimes rendered difficult by the fact that tubercle may occasionally affect several joints, and thus completely resemble a non-tubercular chronic arthritis. Unless some typical sign (osseous focus, sequestrum, abscess) occurs in one joint, the case may remain obscure for years, and even for a lifetime.

Perthes has shown that children may suffer from hip disease with deformity, which has nothing to do with tubercle, although the skiagram suggests it, by revealing gradual destruction of the upper part of the head of the bone. In these cases, however, a thin layer of cartilage persists, and the impairment of movement is very slight, both of these signs being significant of the condition which is illustrated in fig. 468.

CHAPTER XCVII.

SWELLINGS AND TUMOURS OF THE THIGH.

IF we see a patient who has suffered a severe injury, lying with his thigh abducted and the knee slightly flexed, and the whole limb in a position of complete eversion, there is only one diagnosis possible, fracture of the shaft of the femur; if, on closer observation, we see that the thigh is thickened and its axis slightly bent (fig. 469) all doubt is dispelled.

If a little child, who can give us no history, and whose mother does not know whether it has had a fall, presents a swelling of the thigh and cries when touched, avoiding any movement with the leg, the question of fracture or of the early stage of acute osteomyelitis will at once pass through our mind (fig. 471). But we should be relieved from doubt by finding either false mobility on the one hand, or general and local rise in temperature on the other hand.

If the swelling is only slight, but bilateral, the case is neither fracture nor osteomyelitis, but is probably one of **Barlow's disease,** in which very painful subperiosteal effusions of blood occur over the femur. This diagnosis is verified by dark bluish swelling of the gums where the teeth have erupted.

If a young person becomes ill with high fever and severe pain in the thigh, and diffuse swelling thereof occurs within a few days, the diagnosis of acute osteomyelitis is clear.

We shall refer to its various stages in connection with osteomyelitis of the tibia. A comparison of fig. 469 with fig. 470 shows how closely the position of the femur in osteomyelitis resembles the position in fracture.

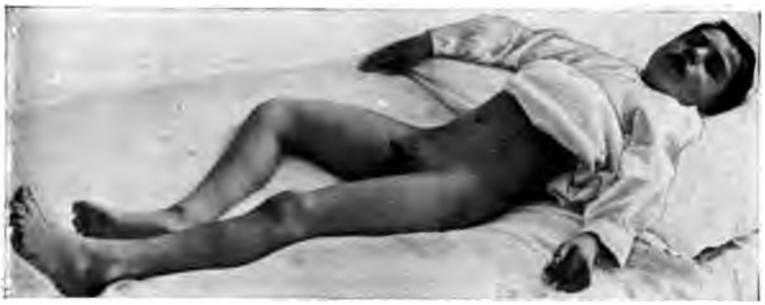


FIG. 469.—Fracture of the shaft of the right femur.

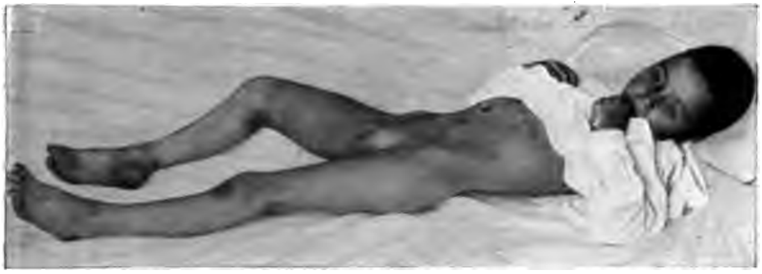


FIG. 470.—Acute osteomyelitis of the right femur.

Swellings which come on gradually and without fever are not necessarily cold abscesses or new growths, but may be chronic forms of staphylo- or streptomycosis of the bone. In order to distinguish between the various possibilities we must decide whether the swelling arises from the soft tissues or from the bone. This can only be determined by its degree of mobility in relation to the bone when the muscles are completely relaxed. As the swellings are not, as a rule, painful, this examination can usually be made without an anæsthetic. If a sarcoma, originating in the soft tissues, has once become adherent to bone, differentiation is no longer possible. The history may, however, indicate that the swelling was movable at first.

A.—SWELLINGS OF THE SOFT TISSUES.

(1) The *skin and subcutaneous tissue* may be the seat of soft fibroma—fibroma molluscum—lipoma, especially in the upper part, lymphangioma, cavernous angioma, varicosities and occasionally sarcoma, originating in a nævus. Their differential diagnosis is too easy to require any consideration here.

(2) *Swellings of the deeper soft tissues* may arise in the lymphatic glands, blood-vessels, muscles, aponeurosis, nerves, and the loose connective tissue.

(*a*) *Swellings of the Lymphatic Glands.*—The crural glands, which alone concern us here, receive lymph from the whole of the lower limb, including the contiguous region of the perinæum. Any swelling in this region demands a search for some portal of entry of infection before we think of anything unusual.

A young man came to the out-patient department with a small swelling in the crural region. The swelling pulsated, or at any rate appeared to do so. The recently qualified assistant, therefore, diagnosed an aneurism. It was, however, an inflamed gland over the femoral artery, and a small septic wound of the skin of a toe was found.

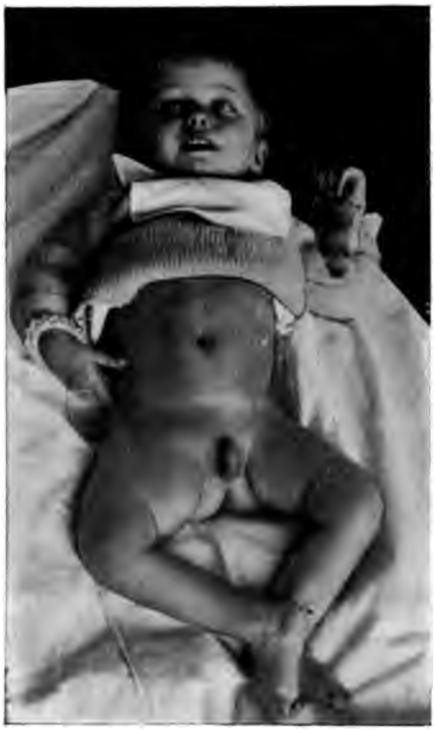


FIG. 471.—Acute osteo-myelitis of the right thigh.

We shall refer to sarcomata of the lymphatic glands later on.

(*b*) *Aneurisms.*—The diagnosis of aneurism is easily made by its position in the course of a large vessel, almost always the femoral artery, and by its pulsation, as also by the vascular conditions below the tumour, and, finally, by its frequent traumatic origin. Not every pulsating tumour is, however, an aneurism, for there are some very vascular sarcomata which pulsate, and over which a distinct murmur is heard on auscultation. It is therefore necessary to employ every expedient to verify the diagnosis of aneurism, especially the compression of the femoral artery just above the swelling. *Arterio-*

venous aneurisms may also occur in the thigh, but their diagnosis is facilitated by the fact that their origin is always traumatic.

False aneurisms caused by bullet wounds sometimes manifest themselves merely by an increasing swelling, induration and heat over the soft parts involved, without any definite pulsation. Surgeons frequently mistake them for abscesses, and the error is only discovered when the blood gushes forth after incision.

(*c*) *Muscular Swellings.*—**Angiomata, tubercular and gummatous nodules, herniæ and osteomata of muscle** behave as in the upper limb. It may be stated, however, that herniæ as well as bony nodules, are mostly found in the adductor muscles of riders, because they are subject to great strain ("rider's bone"). Such a bony formation may also follow a single injury, for instance, a muscular contusion due to the kick of a horse. I have also seen it follow rupture of muscle through over-extension.

There has been much controversy as to the possibility of ossification within a muscle, independently of injury to the periosteum and misplacement thereof. In my opinion there is no doubt about this possibility, although it happens that in the thigh the periosteal origin of such traumatic muscular osteomata has been demonstrated in many cases.

We will discuss sarcomata together with tumours of the connective tissue.

(*d*) *Connective Tissue.*—These tumours consist of **fibromata** and **sarcomata.** They may arise from the connective tissue between the muscles and nerves or within them. A firm tumour, which remains movable for years and causes no disturbance beyond its size, is a fibroma. The more rapid the growth of a tumour, the sooner it contracts adhesions, the more pain it excites, and the richer it is in nucleated cells, the more it approximates to the type of sarcoma. As no sharp limitations can be drawn, even histologically, a positive diagnosis cannot be made clinically. It is better to eradicate the tumour before it becomes malignant than to wait until a positive diagnosis is possible.

There are some fibromata, which at first recur as such, but in course of time their histological type approximates to that of sarcoma. It is therefore necessary to remove even the most innocent tumour while the patient is still in good health.

The relations of the tumour to the muscles may be ascertained by examining it when the muscles are relaxed and when they are tense and comparing the results. A **neuro-fibroma** is diagnosed by its position along the course of a nerve, by its spindle or cylindrical shape, by the early onset of neuralgia and paræsthesia, and occasionally by its multiplicity or the existence of similar tumours in other portions of the body. The risk of secondary malignant degeneration is always present in these cases.

B.—SWELLINGS OF THE BONE.

(1) OSTEOMA AND CHONDROMA.

If we find close to the lower epiphyseal line a nodular bony tumour which has existed for some time, and which is gradually growing away from the joint, we have before us the classical picture of a **cartilaginous exostosis** (figs. 472 and 473).

These growths are congenital and arise from misplaced fragments of cartilage: they consist of bony tissue covered over by a thin layer of cartilage, and they continue to grow until the bone to which they belong has continued its growth. As they are usually situated on the diaphyseal side of the epiphysis, they become more and more distant from this line as the bone grows in length. Sometimes these exostoses occur in separate attacks, and we may therefore find several on the same bone, at various distances from the joint. Sometimes they drag with them an extremity of the joint capsule with which they have contracted adhesions in their original position; in other cases they are covered by a mucous bursa, quite independent of the joint—*exostosis bursata.* If the diagnosis still remains uncertain despite the consideration of all these points, we should examine the other epiphyses of the body and probably find similar exotoses in other positions.

Pure cartilaginous tumours, which may also exist at some distance from the epiphyseal line, form the transition between these innocent tumours and malignant growths of the femur. If they are accessible to palpation, they present the well-known nodulated surface and a consistence less hard than osteomata. Central chondromata, are not, as a rule, diagnosed, until they lead to spontaneous fracture.

(2) SARCOMATA AND ALLIED TUMOURS.

We must never diagnose a primary malignant growth of the femur, before assuring ourselves that the case is not one of secondary growth. This is more especially necessary in connection with the upper half of the femur, because it is a favourite situation for such metastases. Cancer of the breast is the most likely original source, but the primary disease may be cancer or sarcoma anywhere.

I have seen fracture in the upper third of both femora occur in an aged female after cancer of the breast. The accident was merely a slight slip.

In the absence of such an origin, we may assume that a new growth is present—probably some variety of sarcoma.

The so-called *blood-cysts of the long bones*, some of which were formerly termed *aneurisms of bone*, are of a doubtful nature ; at any rate they are not obviously sarcomata. A spindle-shaped distension of the diaphysis, looking like a large beetroot, takes place. The cortical portion of the bone is converted into a thin shell, and its interior is occupied by trabeculæ. The structure contains pure blood. There are no tumour elements visible; but it may be possible to separate from the internal surface a thin layer of tissue containing elements similar to bone marrow, especially giant cells. But whether the condition is a tumour, an inflammatory process ("ostitis fibrosa"), or the result of an injury (an abnormality of callus) is a subject of controversy (see also under "Leg"). Attention is usually first drawn to this condition in the femur by a spontaneous fracture.

In other cases these growths are rather of *connective tissue* nature, and they cause bending, before spontaneous fracture occurs. Growths of this kind have been described as occurring in the subtrochanteric region.

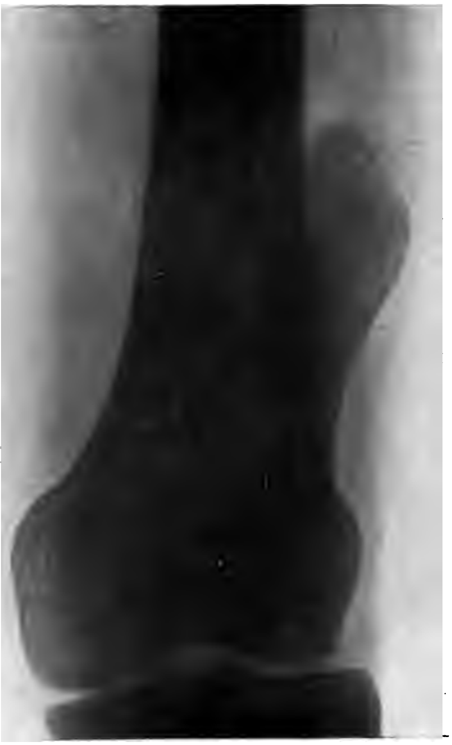


FIG. 472.—Cartilaginous exostosis of femur.

Putting aside these conditions, which are rarities, sarcoma of the femur presents itself either as a diffuse thickening of the epiphyseal region or as a spindle-shaped distension of the diaphysis, which, later on, becomes nodular in character. For the purpose of discussing the differential diagnosis, we must separate these two forms, and we shall begin with the tumours of the *epiphysis*.

(a) Tumours of the Epiphysis.

Sarcoma of the upper end of the epiphysis is usually treated as a form of hip disease, or as sciatica, or even as osteomyelitis, until the neck of the femur suddenly breaks, or the skiagram reveals the existence of something which is neither an ordinary inflammation of the hip-joint nor a simple neuralgia.

I once saw a young man who was of an age when osteomyelitis is frequent, with a high temperature and a rapid pulse. There was at the upper end of the thigh a soft elastic swelling which any clinical student would have considered fluctuating. I also thought that the swelling probably contained fluid, and I made an exploratory puncture in order to decide between tubercle and osteomyelitis. The examination of the tissue thus obtained, and the exploratory incision itself led to the conclusion that there was a small round-celled sarcoma of gelatinous appearance invading the pelvis. This was before the time of Röntgen rays and the young man rapidly succumbed.

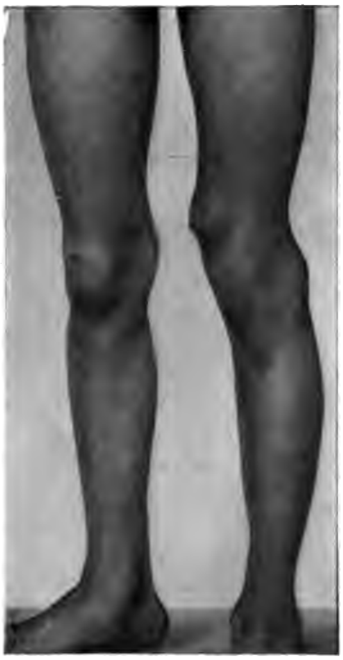


FIG. 473. — Cartilaginous exostosis of the left femur.

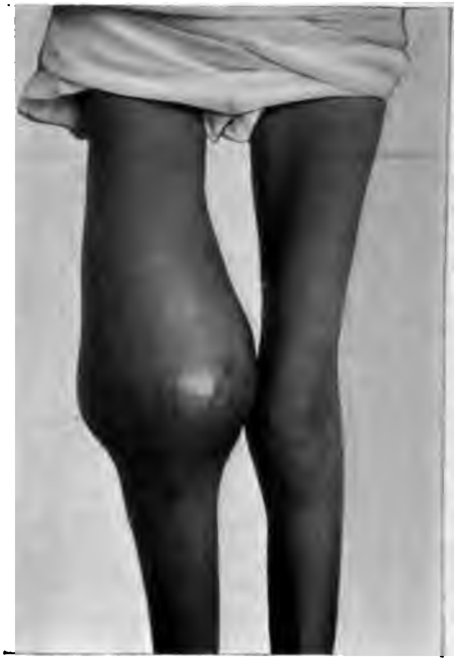


FIG. 474.—Sarcoma of the lower end of the femur.

A vigorous man had kept his bed for several months because of "sciatica." On measuring, it was found that there was supratrochanteric shortening, and the skiagram showed distension of the trochanter by a growth, with the neck of the femur wedged into it.

Sarcoma at the lower end of the femur is easily mistaken, at first, for a tubercular joint.

A middle-aged man noticed a slight swelling about the knee, after a blow. A very experienced practitioner made the diagnosis of tubercular knee, and a surgeon who was called into consultation agreed.

Five years went by, with iodoform injections and waiting. The knee continued to swell, but the movements of the joint remained free. Finally, the lower end of the femur assumed the appearance of a club-shaped structure, about as big as two fists. It was evidently a case of giant-celled sarcoma, covered by a thin shell of bone, similar to the case illustrated in fig. 474, which was also first diagnosed as tubercle.

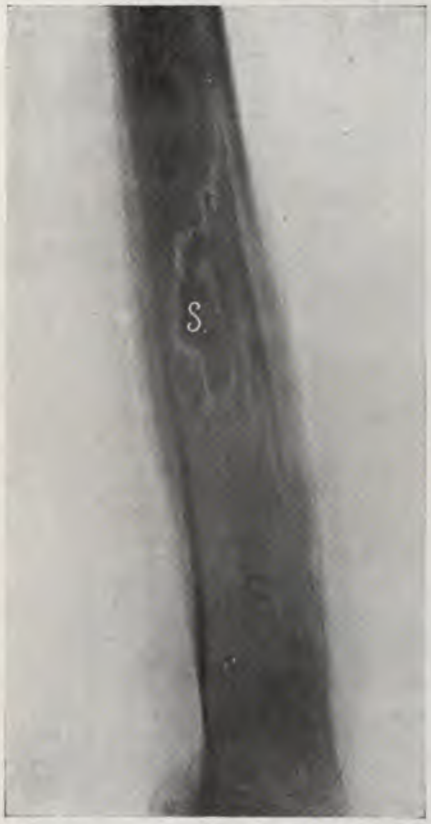


FIG. 475.—Osteomyelitis of the femur, of a few months' duration. Periosteal bone formation beginning. S = sequestrum.

FIG. 476.—Old osteomyelitis of the femur.

The main point in diagnosing these cases is to localize the swelling accurately. In tubercle the *capsule* is thickened, but the underlying *bone* is of normal dimensions. The thickness of the capsule is easily detected by comparing both knees on palpation, especially at the borders. If the whole region of the knee appears to be swollen, although the soft tissues and the capsule are free, we must assume the existence of a more deeply situated process—either a tumour of

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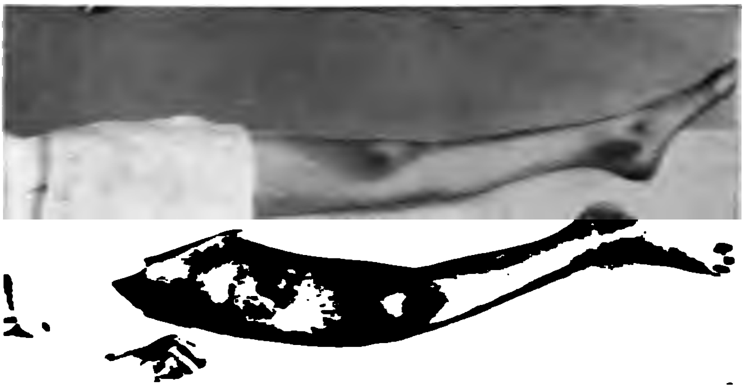


[illegible] of the lower end of the diaphysis.

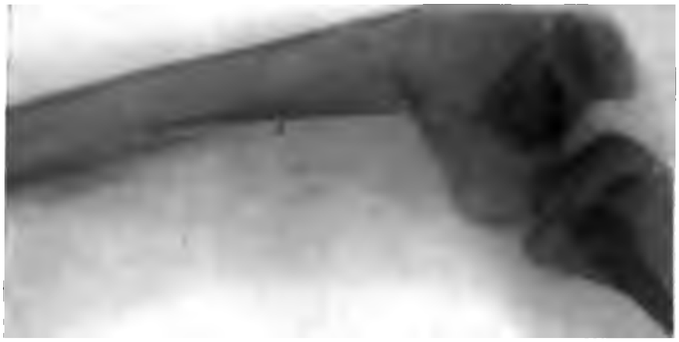


Fig. 478. Skiagram of fig. 477. *S* = sequestrum.

Chronic osteomyelitis is more likely to be a source of error than tubercle. If the growth is characterized by sudden exacerbations, and especially if there are periods of deep and throbbing pain in the bone, accompanied by acute transitory effusion into the joint, the case is probably chronic osteomyelitis. If the disease has persisted for many years, we must assume that an inflammatory process exists. In the case just quoted there was a duration of five years; but this is the extreme limit which is consistent with the diagnosis of sarcoma. As a rule, the course of these tumours permits the diagnosis to be made very much earlier than this.

The appearance of a definite subcutaneous *venous network* is another sign which should be mentioned as an indication that the deeply situated large veins are compressed. This rarely occurs in tubercle, but may take place in osteomyelitis if thick periosteal induration form. It occurs most frequently, however, in connection with malignant growths.

(b) Tumours of the Diaphysis.

Tumours of the *diaphysis* are more difficult to diagnose than those of the epiphysis, because they are less accessible to palpation, and because the characteristic changes in the knee-joint are less in evidence. Otherwise the rules applicable to the epiphysis apply here also. It should be especially noted that osteomyelitis has a much greater tendency than sarcoma to travel along the whole extent of the shaft of the femur. Cases wherein the tumour is sharply delimited from the shaft of the femur are therefore easy to recognize; difficulty of diagnosis arises in connection with the more diffuse, spindle-shaped sarcomata.

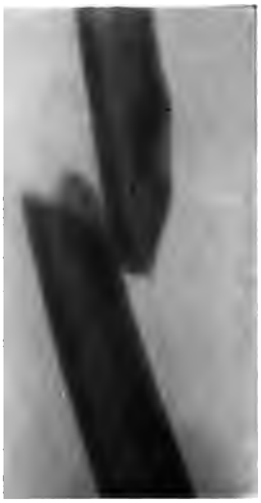


FIG. 479.—Fracture of healthy shaft of femur.

Two examples will illustrate how errors of diagnosis may be caused by the unusual behaviour of the staphylococcus.

A man, aged 32, otherwise in good health, had been suffering pain in his right thigh for a few weeks. The pain did not come on suddenly, and the patient was not conscious of any antecedent febrile disease, nor had there been any previous injury. For some time the patient thought he had rheumatism, but eventually consulted a doctor, who found a slight thickening in the middle of the femur, and thought of sarcoma. On palpation, however, the structure was somewhat more tender than one would expect in the case of a tumour. The X-rays revealed a slight thickening of the cortex, indicated by a sharply defined shadow. This pointed to an inflammatory condition. As tubercle practically never occurs in the shaft of the femur, the diagnosis was evidently a very mild form of "acute osteomyelitis," or rather a chronic staphylomycosis, which better describes the actual condition present. But even this designation was exposed to the risk of error, because an identical clinical picture can be produced by the streptococcus and other pus organisms. The operation, however, showed that staphylomycosis was correct, because the small amount of pus which was found yielded the *Staphylococcus aureus.*

I saw the other case as a student in Kocher's clinic. It was subsequently recorded by Kocher and Tavel in their work on staphylomycoses. The tumour was situated in the lower portion of the femur, and completely simulated a sarcoma. It contained a brownish yellow granulation-like tissue, under a thick indurated sheath. A piece of the tissue was examined by a competent histologist during the operation, and declared to be sarcoma. Amputation would have been resorted to had not a small sequestrum which was found deep down, indicated another diagnosis. Inoculation on a nutrient medium yielded the *Staphylococcus aureus*, and the leg was saved.

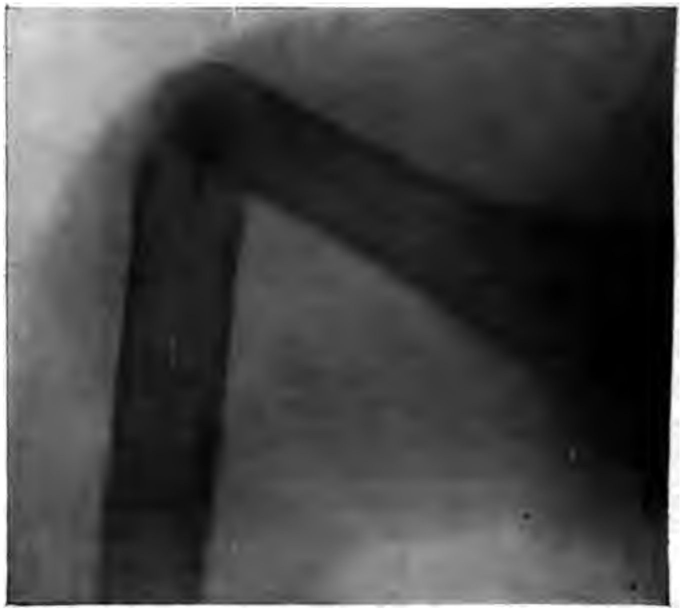


FIG. 480.—Spontaneous fracture in myelogenous scarcoma.

Spontaneous bending of the bone sometimes occurs as a result of osteomyelitis, and is due to the traction of the flexors of the knee on the partially destroyed diaphysis. Figs. 477 and 478 illustrate the consequent typical deformity. If there is a fistula, as there was in this case, the diagnosis cannot be missed.

Sometimes—but not always—the *skiagram* differentiates between osteomyelitis and sarcoma. The following applies both to the epiphysis and the diaphysis.

The skiagram of osteomyelitis shows either (1) a normal edge to the bone; or (2) sharply defined, often clearly laminated deposits (figs. 475 and 476), gradually shelving away on both sides. The former appearance means that the process is comparatively recent, and that the palpable swelling depends upon periostitis without new bone formation. The latter appearance means that the process is somewhat old, and has led to periosteal bone formation. The limits of the sequestrum (figs. 475 and 478) may often be recognized at this stage. The older the process, the less clear is the lamination, so that finally the appearance is that of a diffuse spindle-shaped or cylindrical thickening of the bone (partially in fig. 476).

In myelogenous sarcoma the borders of the femur are equally enlarged, or on one side only. The cortex of the distended portion of the bone is greatly thinned—it may be as thin as paper. The osseous structure is obliterated, and light patches appear in the bone. Spontaneous fractures caused by malignant growths are remarkable, as seen in the skiagram, by the rounded ends of the fragments or by the peculiar erosion of the cortex (*cf.* figs. 480 and 481). Periosteal sarcoma appears as a deposit, on the normal or more or less deeply eroded bone, which casts a light shadow. Sometimes pronounced deposits of periosteal bone appear, which make the picture very similar to that of osteomyelitis.

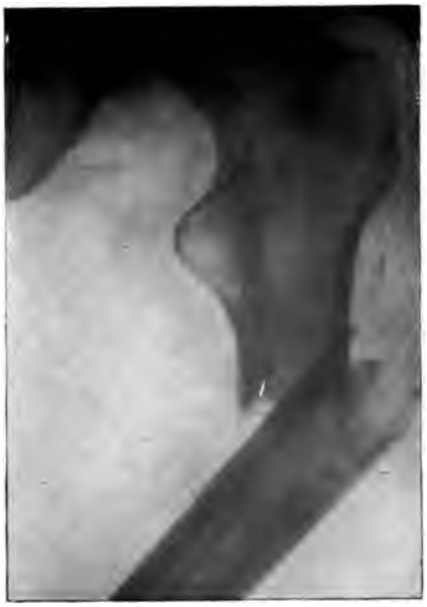


FIG. 481.—Spontaneous fracture in a metastasis of cancer of breast.

CHAPTER XCVIII.

INJURIES IN THE VICINITY OF THE KNEE-JOINT.

In falling forward we instinctively bend the upper part of the body backwards, in order to protect it—and especially the face—from damage. The force of the fall is therefore born by the knee, and by the hands which are extended at the same time. This explains the great frequency of injuries to the knee-joint.

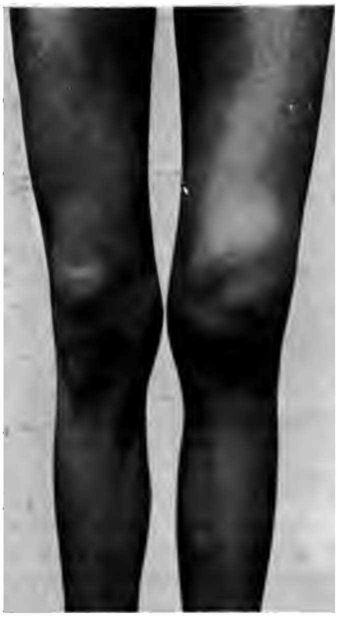


FIG. 482.—Effusion into the joint in sprained knee.

(1) If we find a swelling in front of the knee, after a fall directly upon the joint, and if on palpating the patellar region a cushion-like feeling is obtained, there is an **effusion into the pre-patellar bursa.** The more quickly the swelling develops after the fall, the more likely is it to contain blood; the longer its development is delayed, the more likely is it to be a *serous effusion*, i.e., *pre-patellar bursitis.*

(2) It is more frequent to find some interference with movement — limitation of flexion—and a swelling not localized to the *front* of the patella, but *around* it, the groove on either side of it being obliterated (fig. 482). At the same time a transverse swelling appears above the patella, especially when the quadriceps tendon is relaxed. Sometimes the obliterated lateral grooves become converted into genuine swellings, and the superior transverse swelling increases in size. The patella is raised, and although it may be pressed back against the condyles of the femur, it instantaneously returns to its former level—so-called "riding" of the patella. The French term "ballottement" is, however, more accurate. Such a condition indicates an effusion within the joint; whether it is more of a serous or of a hæmorrhagic character, depends upon the rapidity of its development. If the skin presents abrasions, in evidence of the *direct effect* of the force, and if the parts upon which the force fell—the

patella and tuberosities of the tibia—are tender, while the region of the lateral ligaments is free, the diagnosis is **contusion** of the knee-joint.

If the injury was indirect, taking the form of excessive adduction or abduction or rotatory movement, we must assume the existence of a **sprain**, as long as the symptoms are limited to effusion and interference with movement.

The manner in which the accident happened often makes it impossible for us to distinguish between contusion and sprain. The patient, for example, has fallen down, and lights upon his leg in a constrained attitude—more or less in the "tailor's attitude." He may have sustained a contusion or a sprain. The absence of cutaneous abrasions does not necessarily exclude the latter. We therefore adopt another sign to differentiate between these two injuries: the localization of the tenderness to pressure. We have already seen where this is in cases of contusion. In sprains, however, it is found in the vicinity of the lateral ligaments, because the main force of the injury is situated there. Sometimes the tenderness exists over both lateral ligaments, or over their attachments to the femur and tibia; sometimes only over one ligament—generally the inner.

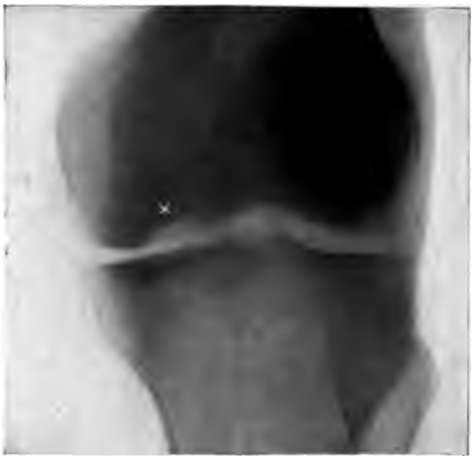


FIG. 483.—Fracture of cartilage over median condyle of femur (x).

It may be objected that these points are unnecessary refinements, seeing that the treatment is identical. It should, however, be remembered that contusions and sprains have their special complications, which will be indistinguishable clinically unless the nature of the original injury is recognized. In both cases, it sometimes happens that the trouble does not subside in the usual way. Creaking sounds, which are often audible at a distance, may occur in the joint; sudden severe pains interfering with movements, or a rapidly developing effusion may arise—conditions which were previously described by the term **"derangement interne."** This vague diagnosis was rendered necessary by the vague knowledge of the actual conditions within the joint, in pre-antiseptic days, when operation was justifiably avoided. But to day we possess data which enable a more accurate diagnosis to be made. If the original injury was a contusion, the internal lesion consists of the fracture of the cartilage of one of the condyles of the femur—generally the median. The detached piece has hyaline cartilage on the one side, and more or less altered bone on the other side. The depression, covered by a smooth cartilage-like scar, may be found, years afterwards, on the condyle (fig. 483). These fractures have nothing to do with detachment of the insertion of the posterior crucial ligament into the internal condyle.

Experience shows that these fractures are more likely to happen if the cartilage or bone be diseased, e.g., osteochondritis dissecans as described by König. One of our patients, in the course of one year, sustained two fractures of considerable portions of bone and cartilage, although he was quite unaware of the injuries which caused them. He suffered no inconvenience until secondary symptoms set in.

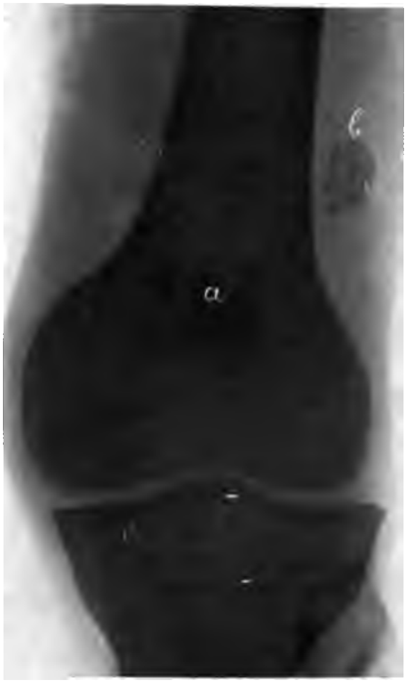


Fig. 484.—Knee joint with two free foreign bodies (*a* and *b*). Patient aged 38.

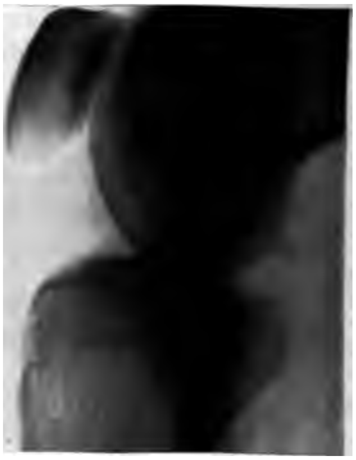


Fig. 485.—Sesamoid (*s*) bone in popliteal space.

It is noteworthy that cretinism—even in its mild forms—predisposes to fractures of cartilage and the occurrence of loose bodies in joints.

The principal evidence of the presence of a foreign body in the joint is derived from actually feeling it, and from an accurate knowledge of the direction of the injury. Repeated examinations may be required in order to palpate the foreign body, and even then one may fail. X-ray examination is conclusive, if some bone is attached to the cartilage, as is usually the case.

A sesamoid bone (fig. 485) in the flexor tendons of the knee must not be mistaken for a foreign body in the joint.

There are some foreign bodies in joints which do not arise from injury.

If the injury was a sprain (generally an eversion of the femur on the fixed tibia), the "dérangement interne" consists of the **detachment** and **displacement** and temporary **"locking" of a semilunar cartilage,** especially the internal. Besides the characteristic pain associated with this lesion, there are two other symptoms which lead to the diagnosis : (1) Pain on pressure over the attachment of the corresponding lateral ligament, or over the attachment of the semilunar cartilage to the tibia ; and (2) the extrusion of the cartilage from the joint cavity on extension of the limb—a somewhat rare symptom.

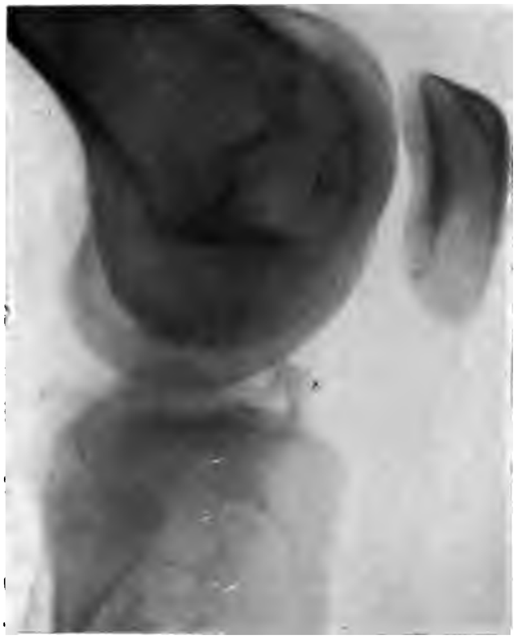


FIG. 486.—Sprained knee with detachment of the insertion of crucial ligament into the tibia (x).

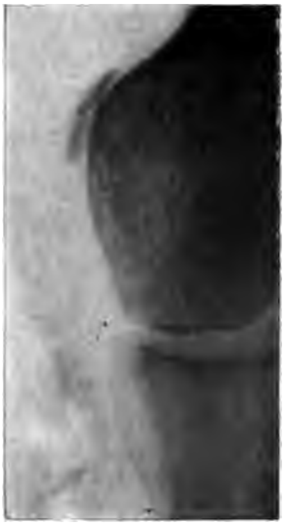


FIG. 487.—Fragment detached from internal condyle of femur.

The former symptom is only found in comparatively recent cases, and the latter is frequently entirely absent, so that we often have to rely exclusively on the history. The differential diagnosis between fracture of the articular cartilage and laceration of the semilunar cartilage may also be suggested by the frequency of the attacks. If these are infrequent, but accompanied by very severe disturbances within the joint, the case is probably one of fracture of the cartilage over one of the condyles. If the attacks are frequently repeated, and if loud grating occurs on any extensive movement, the case is probably a laceration of the semilunar cartilage. But the former symptoms may nevertheless be due to laceration or detachment of the semilunar cartilage. In the quiet intervals of these occurrences, so little may be found on examination that the patient is apt to be suspected of exaggerating or malingering. In spite of typical symptoms, operation occasionally reveals nothing but proliferating swollen folds of the ligamentum mucosum, hanging like polypi, in the joint (Hoffa).

Stieda has described a lamella of bone at the upper end of the internal lateral ligament as a late sequela of sprains.

It is not clear whether this is due to a detachment of periosteum and bone at the moment of the injury, or whether it is due to secondary bone formation in the torn ligament. Both causes may possibly contribute. I have observed the entire disappearance of this lamella within a few months.

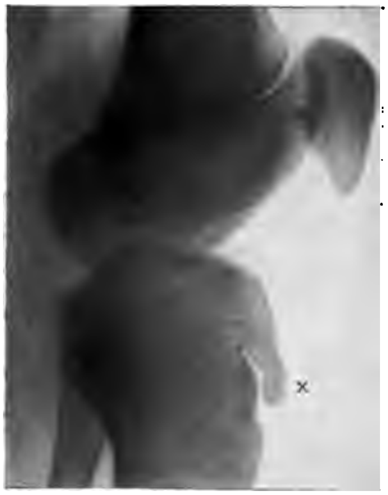


Fig. 488.—Normal spine of tibia in a young person.

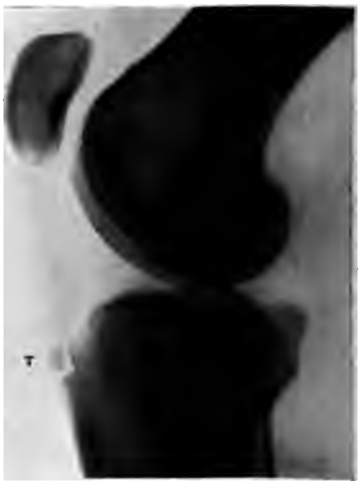


Fig. 489.—Detachment of spine of tibia.

To return to the recently inflicted injury. A patient suffering from a sprain or contusion can lift his leg in a position of extension, although it may cause pain. If this is impossible, we must conclude that there has been some injury either to the extensor apparatus or to the bone itself.

(3) We can tell in a moment whether there is shortening, or pain on pressure on the axis of the limb, and thus decide as to fracture in the continuity of the bone. In the absence of this, the only other possible condition is *laceration of the extensor apparatus.* This may occur in one of three situations: (1) In the quadriceps tendon above the patella; (2) in the patella itself; and (3) below it, i.e., in the

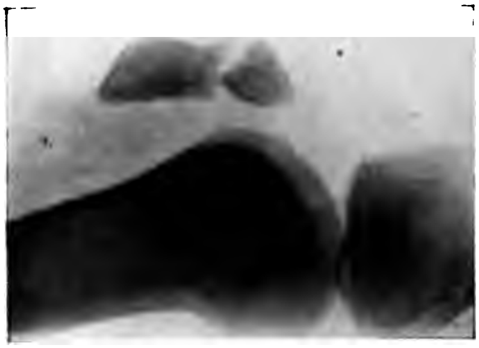


FIG. 490.—Transverse fracture of patella.

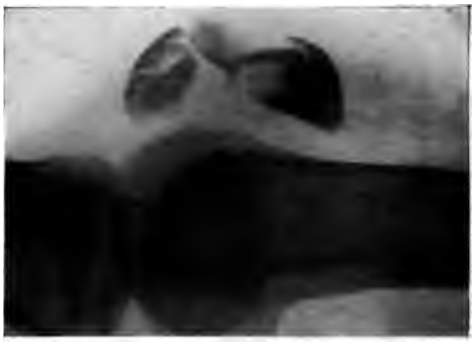


FIG. 491.—Comminuted fracture of patella.

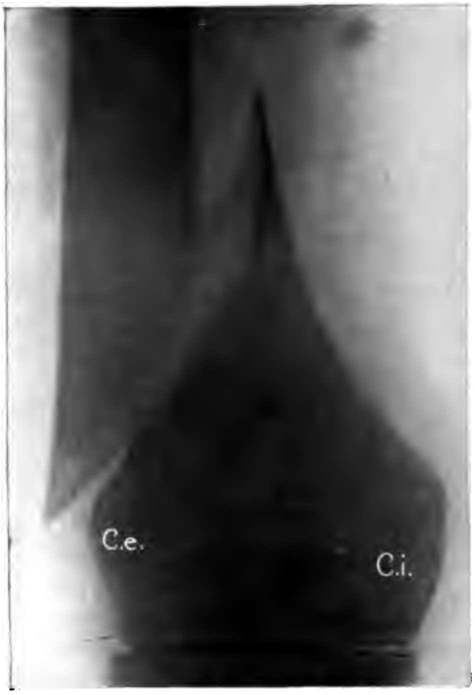


FIG. 492.—Supracondylar fracture, seen from the front, with typical displacement. *C.e.* = external condyle. *C.i.* = internal condyle. Upper fragment displaced forwards and outwards.

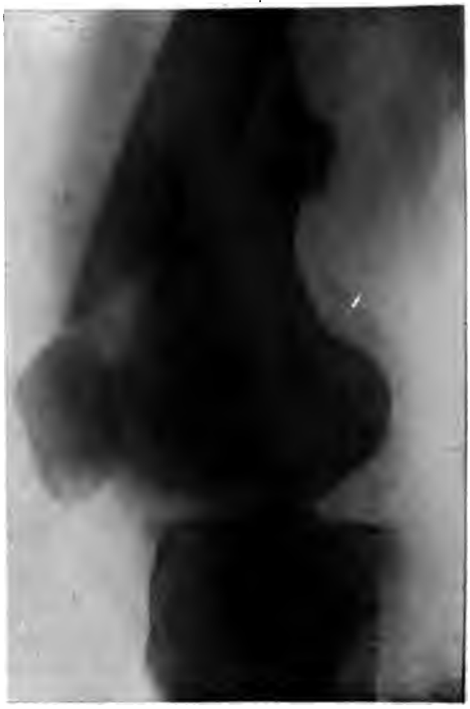


FIG. 493.—Supracondylar fracture, seen from the side. Typical displacement of lower fragment.

ligamentum patellæ. Palpation of the region involved will at once give an approximate idea of the situation of the injury. **Laceration of the quadriceps tendon,** the rarest of these injuries, produces an easily palpable and even visible hollow above the patella. This is especially striking if the upper end of the tendon rolls up, and thus appears to be thickened.

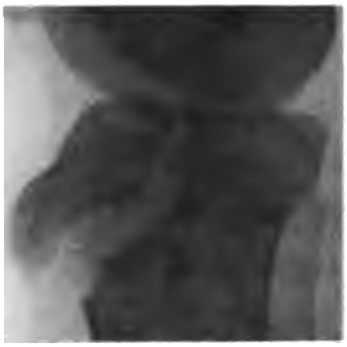


FIG. 494.—Detachment of tuberosity of tibia.

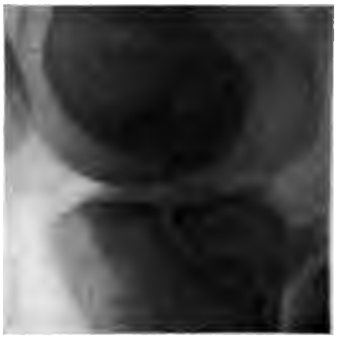


FIG. 495.—Fracture of posterior edge of head of tibia.

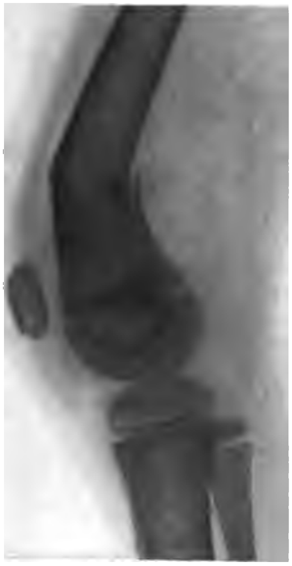


FIG. 496.—Supracondylar fracture. (From a case of cretinism.)

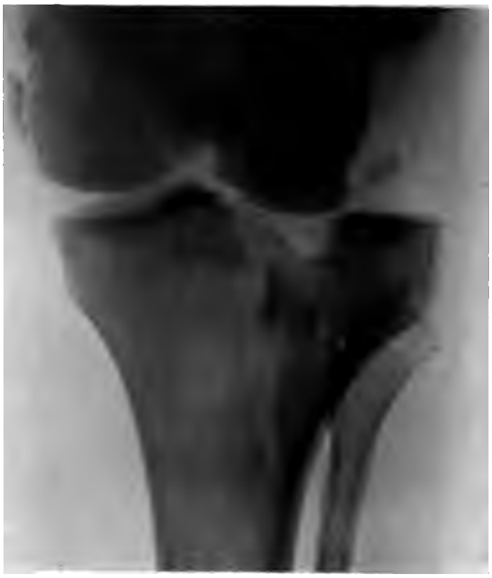


FIG. 497.—Fracture of external tuberosity of the tibia. Fragments torn and broken off the femoral condyles.

Detachment of the ligamentum patellæ close to the knee-cap, with some bone substance adherent to it, is of more frequent occurrence. Both these injuries, especially the former, imply some previous debilitated condition of the tissues. If the age of the patient does not afford a sufficient explanation, we should examine for the presence of syphilis or diabetes. **Fracture of the patella** is the most frequent example of rupture of the extensor apparatus of the knee. The condition is unmistakable.

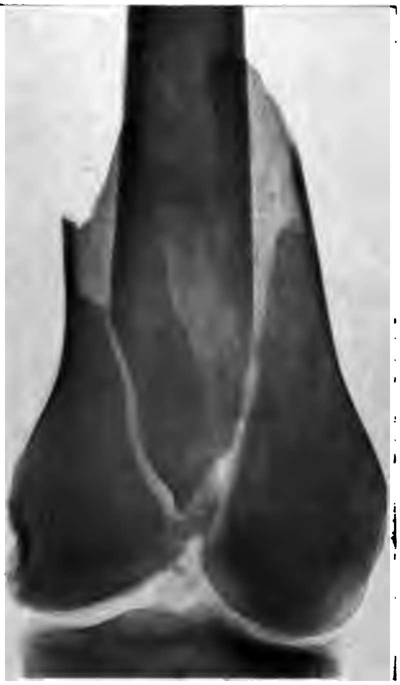


Fig. 498.—Condylar fracture. (*Post-mortem* preparation.) Y-shaped fracture.

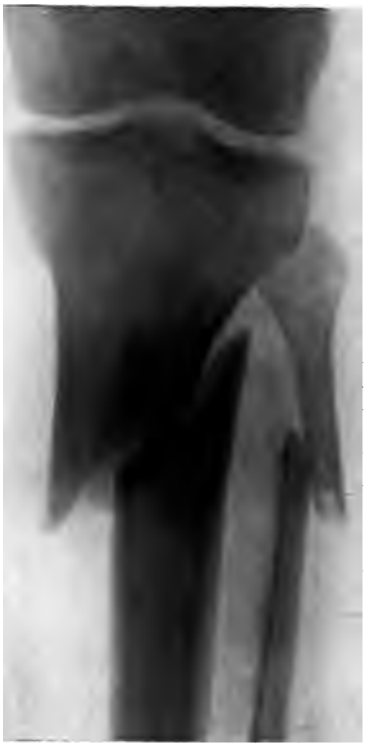


Fig. 499.—Fracture of the tibia, below tuberosities.

The precise manner in which fracture of the patella occurs has been a subject of much controversy. It has been stated that the simple transverse fracture (fig. 490) is indirect, and caused by muscular contraction, and that the Y-shaped or radiate fracture (fig. 491) is caused by direct force, i.e., by falling on the patella. Four-fifths of the cases of fracture of the patella were attributed to muscular action, whereas, as a matter of fact, recent careful observations show that the proportion is exactly the reverse.

Schlatter describes cases wherein the spine of the tibia has been torn off or broken off at the epiphyseal line in young persons (fig. 489). One must, however, guard against mistaking the normal line of the epiphysis, however unusual it may look, for the result of some trauma (fig. 488). In most cases the pains are due to tearing and not to gross anatomical changes.

(4) If, on the contrary, there is pain on axial pressure with shortening, however slight, combined with severe hæmorrhagic effusion into the knee-joint, we conclude that the lesion is not in the extensor apparatus, but that it is located in the *continuity of the limb*, i.e., either a fracture of the lower end of the femur or the upper end of the tibia. These fractures are classified as supracondylar, diacondylar (in the epiphyseal line of the femur), fractures of the external and internal condyle, combined **Y-** and **T-shaped** fractures. In the tibia the fracture may concern one or other or both tuberosities, the anterior or posterior edge of the head of the bone, or it may be below the tuberosities.

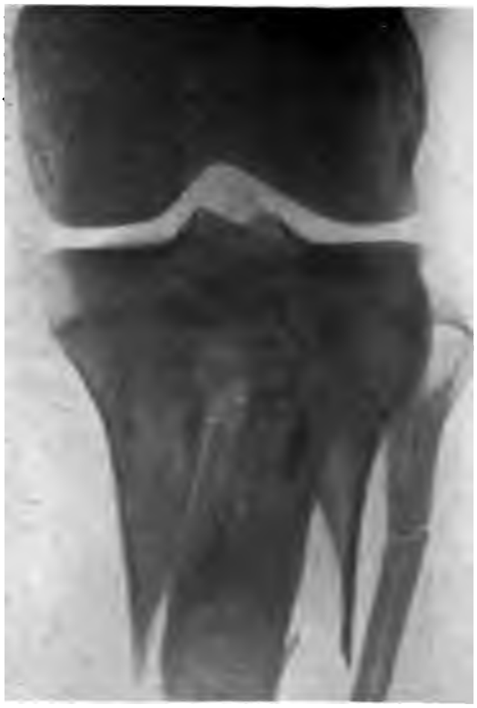


FIG. 500. -Y-shaped fracture of tibia (caused by a wrench), with cracking of fibula.

Supracondylar fracture does not involve the joint directly, but it often happens that the upper fragment, which is usually displaced forwards and outwards, penetrates the joint between the lower fragment and the patella. The joint cavity is thus opened up and participates in the hæmatoma. The usual course of the lines of fracture is seen figs. 492-496.

Fig. 496 represents the case of a dwarfed cretin, whose femora had, for a whole year, undergone subperiosteal bending at the same situations. Hypothyrodism is one of the chief causes of diminished bone stability. I was once consulted for a patient with thyroid inadequacy who had just sustained his twenty-second fracture.

If the hæmorrhagic effusion is considerable, an accurate diagnosis cannot be made without an X-ray examination, which should always be made in two directions at right angles to each other.

Diacondylar fracture, occurring as a separation of the epiphysis, is a very rare event, and if the displacement is slight may be mistaken for a sprain. If the displacement is considerable, or if false mobility exists, the diagnosis is based upon the facts of lateral displaceability and the absence of any interruption of continuity *above* the joint line.

Fracture of the condyles in their various combinations are recognized by the mobility of one or both detached condyles, on the shaft of the femur, and by the varus or valgus position assumed by the joint. **Y-** and **T**-shaped fractures arise from the shaft of the femur being driven in, like a wedge, between the condyles (fig. 498). Details are accurately obtained by a skiagram, which is preferable to the otherwise unavoidable examination under an anæsthetic.

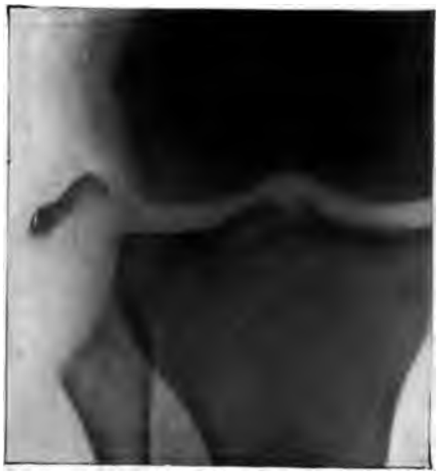


FIG. 501.—Detachment of head of fibula.

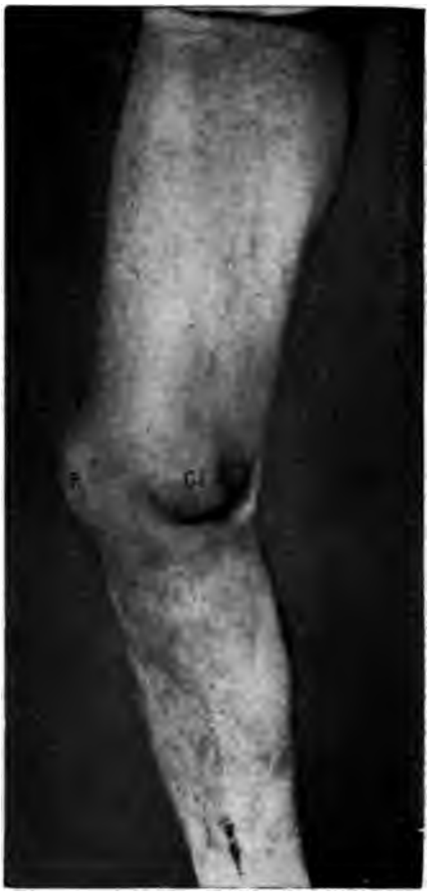


FIG. 502.—Dislocation of the knee. Prominence of the internal condyle. *Ci.*, caught by the buttonhole mechanism ; *P.*, patella.

Corresponding fractures of the head of the tibia are characterized by the slight amount of false mobility of the fragments, so that the diagnosis must be based on the localized tenderness on pressure, the loss of power and occasionally on the manifest deformity. An accurate decision can only be formed by a skiagraphic examination.

Fracture of the head of the fibula (fig. 500) which is very characteristic but rare, and detachment of its upper end should be mentioned here (fig. 501).

Dislocations of the knee-joint and of the patella are still more rare. *Dislocations of the knee*—both congenital and traumatic—have been seen in all directions, forwards, backwards, downwards and outwards. Their appearances are so remarkable that it is unnecessary to discuss their differential diagnosis. But incomplete lateral dislocations require very careful palpation. The button-hole mechanism, whereby one of the condyles becomes "caught," as first described by Iselin, is worth noting. It is illustrated in fig. 502.

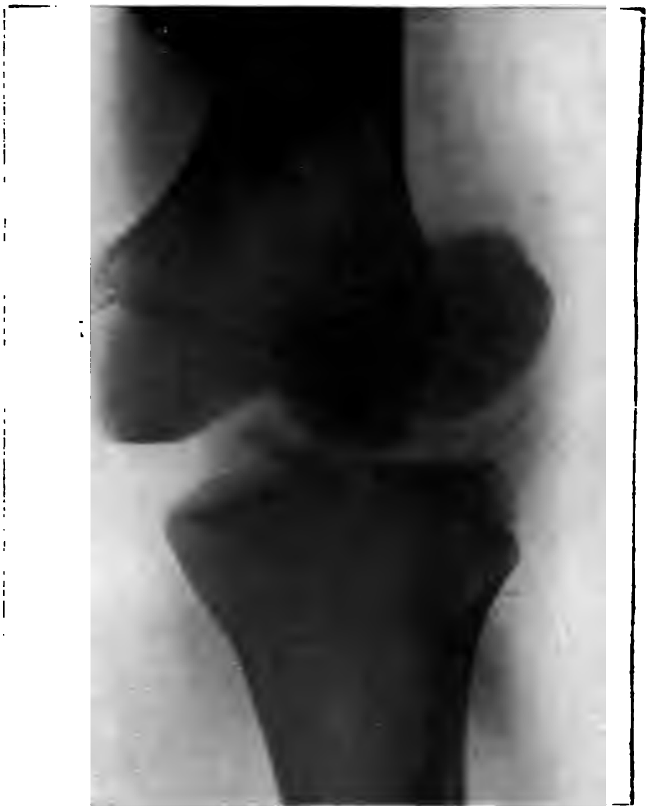


FIG. 503.—Skiagram of same case. Attachments of crucial ligaments to tibia, torn off.

Displacements of the *patella*, which are usually external, are diagnosed by careful palpation. This bone is quite superficial, and there is no difficulty in detecting whether it is displaced outwards or inwards, or perched on its edge. Two cases of vertical torsion through an angle of 180° have been described, but the diagnosis of this condition would be more difficult.

CHAPTER XCIX.

ACUTE INFLAMMATORY DISEASES OF KNEE-JOINT.

ACUTE inflammation of the knee-joint rarely presents any diagnostic difficulty. The first point to settle is, that the disease is really in the joint. Superficial examination may lead to a diagnosis of suppuration within the knee, when the condition is really one of **prepatellar phlegmonous bursitis,** if, as frequently happens, many ounces of pus are present and the whole knee appears to be swollen in an unshapely manner. But the distinction is easy if the examination is careful. The swelling is in *front of* the patella in bursitis; in a suppurating knee, however, the prepatellar region is rather flattened out, and the patella itself can be felt just under the skin. In bursitis the popliteal space is free, whereas, in acute arthritis of the knee, it is painful on pressure.

In considering the knee-joint itself, we must be quite clear as to the elements which such an inflammation may comprise, i.e., (1) an effusion of fluid, (2) swelling of the capsule, (3) bony and cartilaginous changes. Often only one of these signs is present, but frequently there are two, and sometimes all three.

Effusion into the joint is recognized by the filling out of the fossæ on either side of the patella, and by the distension of the suprapatellar bursa. If the effusion is considerable the phenomenon of "riding of the patella" appears. If the capsule becomes lax after the subsidence of the effusion, the knee-cap can be displaced, to a remarkable extent, in all directions.

In order to detect any slight degree of *swelling of the capsule,* it is necessary to compare its fold of reflection on both knees. Normally this can just be felt if the patient is not too fat.

Involvement of the bone is often difficult to recognize, unless some striking change in shape is present. A tender area, which is sharply limited, and not at the situation where the capsule is reflected indicates a lesion in the bone.

The *etiology* of **acute inflammation of the knee** is the same as that of acute arthritis elsewhere. We have already discussed this in full in connection with the shoulder-joint, and will only refer here to a few characteristic conditions.

If we are consulted about a case of acute effusion in the joint, we must first inquire whether there has been any recent injury. In the absence of such a cause we must inquire about former injuries, and previous attacks of sudden joint swelling. If we ascertain that such attacks have occurred and that they have been occasionally, accompanied by severe pain which prevented any further movement, we should think of a *foreign body in the joint* and *locking of a semilunar cartilage.* If the effusion occurs periodically, without the signs of a foreign body, we should think of the rare cases of *intermittent hydrops* of the knee, probably of nervous origin.

In the absence of any injury or any previous attacks of a similar character, we should conclude that the case is of an *infective nature,* if the effusion has come on within a few days with severe pain, tension and pyrexia. If the patient is a young man and the knee is the only joint affected, we should ask him *sotto voce* when he had *gonorrhœa.* He will rarely deny the impeachment, but may ask that it should be treated as rheumatism, out of regard "for the old-fashioned idea of his parents."

We should make the same diagnosis if a young woman has "caught cold" on her honeymoon.

Secondary inflammation of the knee has been observed in infants with gonorrhœa ophthalmia, as also in little girls with gonorrhœa.

Insured persons will attribute it to an accident. In some cases this will be pure invention, but it is quite conceivable that a slight sprain may afford the gonocococcus the opportunity of attacking a joint, if the organism already exists in the body.

An acute inflammation of the knee-joint after a *confinement* or a *septic abortion,* presents difficulties of a therapeutic rather than of a diagnostic nature.

Acute arthritis occurs more frequently in the knee than in other joints, as a result of *direct injury,* varying from a needleprick—the needle remaining in the capsule—to the cut of a hatchet. This is not always followed by acute suppurative inflammation with great swelling and high fever. The knee more frequently swells up gradually in the course of a few days, the fever is slight and the periarticular changes are trivial. The shape of the distended articular space is very clearly discernible through the soft parts. As the symptoms are so mild, nothing but a purely serous exudation is expected; but exploratory puncture shows that some turbidity already exists owing to fibrin and pus cells. If the case is treated without delay in an appropriate manner the knee will be saved, but if delay is incurred stiffness will result.

In growing children every case of acute inflammation of the knee should suggest the possibility of *acute osteomyelitis* of one of the adjacent bones. An articular effusion, sometimes purely serous is often the only symptom of a localized diseased area in an epiphysis.

Diagnostic interest may also attach to the *late sequelæ of acute inflammatory knee-joints*. If, despite extensive incision, the temperature remains high and the neighbourhood of the joint remains swollen, we must assume that *periarticular* abscesses have developed, which are most frequently found under the extensor muscles and the patella. Sometimes the joint continues to suppurate despite multiple incisions, and the temperature persists at 100·5° although no periarticular abscesses can be demonstrated. X-rays will show that the interval between the femur and tibia is abnormally narrow, owing to more or less destruction of the cartilage. On exposing the joint the cartilage will be found to be eroded, the underlying bone more or less destroyed, and in young people the epiphysis may have formed a sequestrum.

CHAPTER C.

CHRONIC DISEASES OF THE KNEE.

THERE is no joint which varies so much as the knee in appearance as a result of chronic inflammation from one and the same cause. Great caution must be exercised in diagnosing the cause from the anatomical conditions present.

Thus simple hydrops of the knee may exist in chronic traumatic inflammation, in tubercle, or in neuropathic disease of the joint; ankylosis may exist in chronic articular rheumatism, and in tubercle, &c.

A chronically inflamed knee may present three essentially different conditions, each one of which possesses its own problems of differential diagnosis :—

(1) Chronic articular effusion.
(2) Thickening of the capsule, including the synovial membrane.
(3) Rigidity of the joint.

(1) CHRONIC ARTICULAR EFFUSION.

We have already studied, in connection with acute traumatic effusions, how to recognize an articular effusion. Chronic effusions, however, last longer and attain a larger size, though their degree of tension is not so great.

Certain articular effusions were previously termed *idiopathic*. We now know, however, that, apart from very rare intermittent effusions due to nervous disturbances, that there is no such variety. The more carefully the history is taken, and the more accurate the examination, the more certain is some cause to be discovered.

(*a*) The diagnosis is usually chronic articular rheumatism, if several joints are affected simultaneously or in rapid sequence, and the remarks already made on this malady, when dealing with diseases of the shoulder, should be recalled. In the latter, the most frequent varieties are the adhesive and the destructive caries sicca, whereas in the knee exudative processes predominate. In the neuropathic forms there is a great tendency to proliferative processes. The diagnosis of rheumatism is often merely a refuge of ignorance. It should never be made until all other possibilities have been excluded. An example will illustrare this.

A boy, aged 10, suffering from articular effusion of both knees, was sent into the hospital as a suspected case of tubercle. But as the affection was on both sides, and as there were no other indications, we thought the case was one of chronic rheumatism. One morning, however, we noticed some injection of the eye and slight cloudiness of the cornea, which had supervened since the previous evening. This at once suggested "the sins of the fathers," and it was clear that the serous inflammation of the knees was due to congenital syphilis. The result of mercurial treatment confirmed this diagnosis.

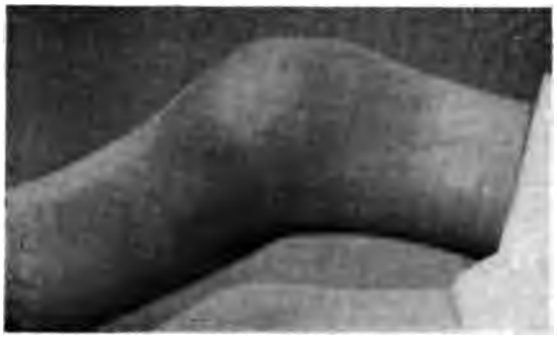


FIG. 504.—Hæmorrhagic effusion into knee joint.

If there has been no attack of recent parenchymatous keratitis, hereditary syphilis may probably be indicated by old corneal opacities or by the shape of the teeth, in addition to the bilateral effusion. In other cases, we may ascertain that the patient bleeds easily, and that any pressure leaves a blue mark. This suggests that an early stage of so-called hæmorrhagic effusion is present—but this is a rare contingency (fig. 504).

We have hitherto been assuming that all the articular diseases are of a serous nature. Although this is true for cases of congenital syphilis, it is not true for hæmorrhagic joints and for genuine chronic rheumatism, in both of which there may be effusion on one side and ankylosis on the other.

(*b*) The problem is quite different if only *one joint* is affected. The most important question is whether *tubercle* is present. In addition, one has to think of (1) chronic or recurrent traumatic effusions, (2) foreign bodies and dislocation of a semilunar cartilage, (3) gonorrhœa with an unusually protracted course, (4) the proximity of a focus of osteomyelitis, (5) tertiary syphilis, (6) rheumatism exceptionally remaining in one joint, (7) a neuropathic joint, and finally, (8) a mono-articular hæmorrhagic effusion. On the other hand, if the effusion is limited to one side, hereditary syphilis may be confidently excluded. We will deal briefly with these possibilities, reserving tubercle till the end.

Traumatic effusions come on in an acute manner. If the trauma is frequently repeated, or if the patient is rheumatic, the effusion may become chronic. There is no evident thickening of the capsule, and this constitutes the distinction from tubercle supervening after an injury. The "locking" of foreign bodies and of the semilunar cartilages, are distinguished by their intermittent character.

A knee, the mechanics of which has been disturbed by a fracture—even extra-articular—may be subject to intermittent and remittent serous effusions, years after the accident.

The diagnosis of **chronic gonorrhœal effusion** is made from the history and usually also from the condition of the urethra.

Synovitis due to osteomyelitis is easy to recognize if the patient has the scars of that disease on the femur or on the tibia. There are, however, cases, which for years are called "rheumatism," when in reality an abscess exists in the bone, close to the epiphysis, and somewhat acute attacks occur from time to time, accompanied by effusion into the joint. We must depend upon the history for the correct diagnosis, but examination will show that the main situation of the swelling and of the pain is not in the joint, but in the adjacent bone.

The diagnosis of **tertiary syphilitic synovitis** is suggested by the history and the slightness of the pain ; but one cannot be certain of its accuracy until specific treatment has met with success. We may assume the presence of chronic **mono-articular rheumatic synovitis** if the capsule is not markedly thickened, the temperature over the joint is not definitely raised, and if the trouble has persisted for years without getting much worse.

Mild forms of tubercle may drag on from infancy far into adult age without abscess formation and with a tolerable amount of movement, but with intermittent exacerbations. In these cases, however, the capsule is always definitely thickened.

In the **neuropathic forms** the diagnosis is established by the absence of pain, despite advance changes in the joint, and by the early onset of deformity, in addition to the pure synovitis.

This variety of arthritis has been appropriately termed a "caricature of ordinary arthritis." If symptoms of tabes or syringomyelia co-exist, the diagnosis is naturally easier; but sometimes it will be necessary to search for these diseases, because the joint trouble may be the first symptoms of nervous disease to attract the attention of the patient.

Hæmorrhagic effusion has already been referred to. We have now arrived, by way of exclusion, to **tubercle**, which is by far the most common form of serous inflammation of the knee. The vast majority of these cases occur in infancy, but adults may become affected, and it is not rare even in old age. It is only distinguished from other forms of serous inflammation by the fact that the capsule is somewhat thickened from the very beginning, and by the persistent elevation of the temperature over the diseased joint. The thickening of the capsule must always be looked for at its *folds*, that is to say, at its superior border, and on both femoral condyles. If the healthy knee is examined at the same time it will be easy to appreciate how far the folds can be palpated in the normal condition.

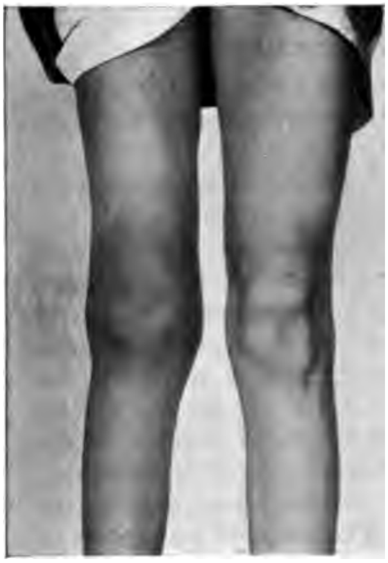


FIG. 505.—Slight tubercular hydrops of the right knee with almost completely free mobility. Very slight muscular atrophy, considering that the disease has lasted nine years.

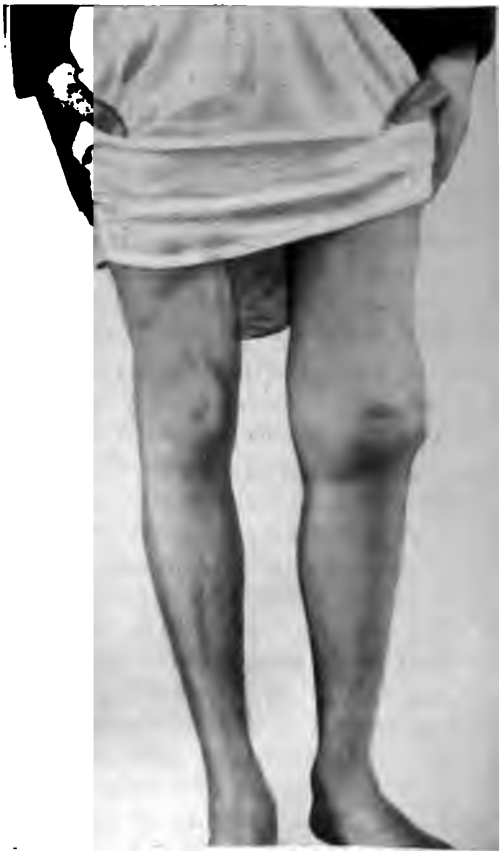


FIG. 506. — Proliferating tubercular disease of knee, with moderate amount of serous effusion. Mobility still partially retained.

If the joint is full and tense, this sign cannot be demonstrated. In such a case, the joint should be *punctured*, and a positive diagnosis will be obtained by examining the fluid. Blood, pure or nearly so, indicates hæmorrhage into the joint. Clear, serous, or mucous fluid may be found in every variety of inflamed knee, but if the fluid is purulent, turbid, or contains shreds of fibrin, the case is probably tubercular.

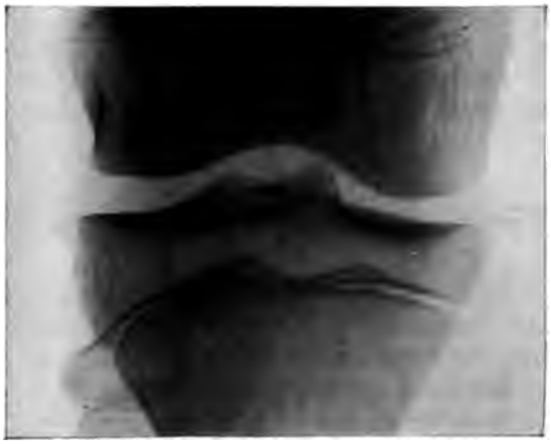


Fig. 507*a*.—Normal knee.

In order to test the temperature, it suffices to lay both hands lightly upon the two equally long exposed knees. Effusions due to gonorrhœa, osteomyelitis and recent injury, show some local elevation of temperature, but this vanishes much more rapidly than in tubercle, wherein the same amount of heat can be detected for many months at each examination.

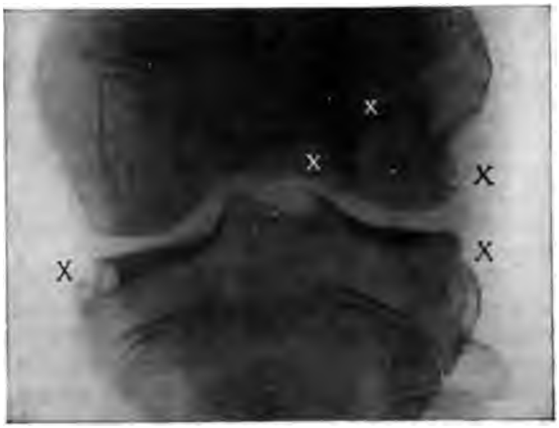


Fig. 507*b*.—Tubercular knee. Secondary erosion of bone at x x. Cartilage somewhat narrowed.

We might expect that tubercular disease of the knee-joint would lead to *early limitation of movements*, as occurs with other joints. But, as a matter of fact, this is very often not the case. The excursions of the knee may remain perfectly free even in tubercular synovitis of many years' standing, as long as the joint is not over distended by the effusion. In such cases the muscular atrophy does not supervene as soon as it does in tubercle with early ankylosis.

We may briefly summarize the above, as follows: Every mono-articular, chronic, serous inflammation of the knee, wherein there is definite thickening of the reflected folds of the capsule, and wherein there is a *persistant definite local elevation of temperature, must be regarded as tubercular, even if mobility still remains free and pronounced muscular atrophy is absent. Nothing but very clear evidence to the contrary warrants us in departing from this rule of diagnosis.*

In rare cases, one or more movable foreign bodies may be felt in the swollen joint. They are not completely free and can only be moved in a small circle. These are examples of the *polypoid form of tubercular knee* (Plate IV, fig. *b*). The polypi consist of hard connective tissue, more or less abundantly permeated by tubercles.

(2) FUNGATING INFLAMMATION OF THE KNEE-JOINT.

If the capsule is greatly thickened in a *diffuse* manner, the case is either tubercle, or the very rare condition of gummatous arthritis, whether there be effusion present or not.

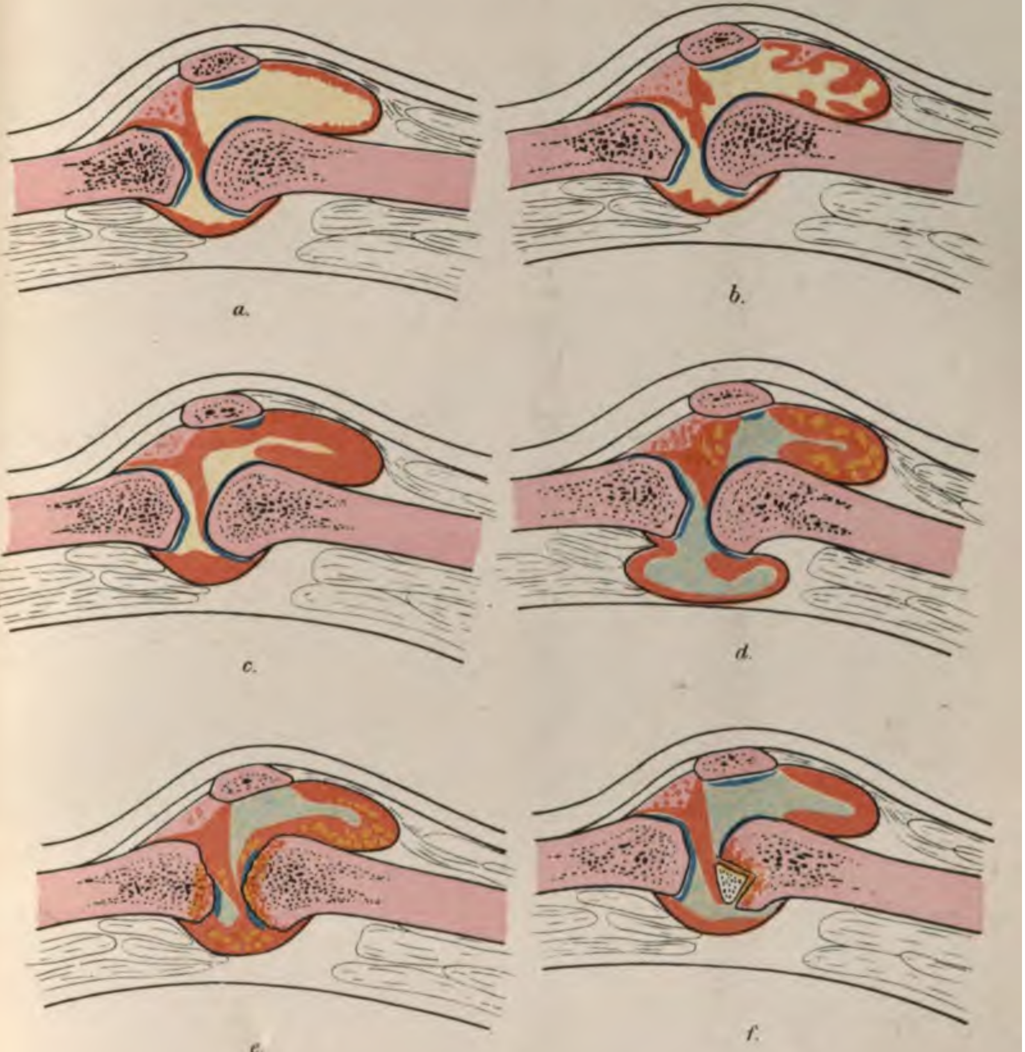
Localized fungating degeneration of the capsule might, apart from gumma, be confused with the rare condition of sarcoma of the articular capsule, if the joint is movable. But as we have already seen, free mobility does not by any means exclude tubercle. In other cases, however, movement is soon interfered with, and finally complete rigidity supervenes.

A momentary glance often suffices to distinguish these two forms, before we ask the patient to make any movements. If the *mobility is preserved* the muscles of the thigh and leg are not strikingly atrophied, and the thickened capsule resembles a moderate amount of effusion, more especially as some effusion usually exists. In cases of *early rigidity*, however, the diffusely swollen knee is slightly bent, the muscles of the thigh and knee are wasted, so that a spindle-shaped appearance results.

The principal question from the diagnostic standpoint is whether the condition is of a *purely fungating* character, with or without serous effusion, or whether it has become *purulent*. We may confidently assume that suppuration has occurred if there appear a circumscribed, elastic, or fluctuating bulging, which seems to be just under the skin. This appearance may present anywhere, but it is most frequent at the level of the joint cleft.

It used to be the practice to incise such swellings early, in order to see their contents, or to remain loyal to the old maxim "*ubi pus, ibi evacua*." As a rule the abscess refused to heal; a fistula developed and secondary infection occurred. In this condition the case was turned over to the surgeon. We now know that such an incision into a tubercular abscess of a bone or a joint, however aseptically performed, almost unavoidably leads to secondary infection by pus organisms and does the patient considerable harm. It is only justifiable to open a tubercular abscess if one is prepared to proceed

Plate 4.



Diagrammatic Representation of various forms of Tubercular disease of the Knee.

Light pink = normal bone
Blue = cartilage
Red = Inflamed Synovial membrane
Light yellow = serous effusion
Green = pus
Orange = Caseous thickening

a. Serous inflammation of Knee. Synovial membrane slightly thickened, invaded by tubercles. Serous effusion.

b. Polypoid inflammation of Knee. On synovial membrane large fibrous polypi with tubercles.

c. Fungating inflammation of Knee. Slight exudation. Much thickening of synovial membrane.

d Fungating and caseating inflammation of Knee. Caseous thickening of fungating masses. Purulent exudation. Abscess behind bend of Knee.

e. The same, but cartilage of bone removed and destroyed by tubercular proliferation.

f. Primary disease of bone, in the form of a wedge shaped area, with sequestrum.

Quervain, Diagnostic Surgery.

There are some rare cases, in which examination leaves one in doubt as between a serous effusion and a fungating thickening of the capsule, and on operation it is found that neither one nor the other exists, but that the condition is one of a lipoma-like proliferation of the articular tufts—so-called **lipoma arborescens.** As this change may occur in chronic arthritis of different origins, and exceptionally also in tubercle, it is not easy to make an accurate diagnosis. I have found this condition limited to the upper segment of the joint.

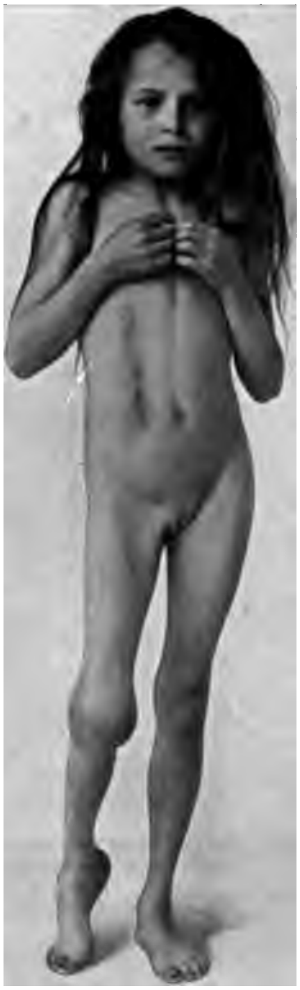


FIG. 510.—Tubercular knee, slightly flexed and contracted, and in valgus position, with subluxation of the tibia backwards and outwards.

(3) RIGIDITY.

If contractures are present, the conditions which come into consideration for diagnosis are again abundant. One must first put aside those cases wherein the history points to some form of acute infective inflammation of the knee-joint, preceding the contracture or ankylosis. We have only to consider rigidity of *gradual* onset, and may even then be in doubt as to the (1) terminal stage of hæmorrhage into the joint, which is rare; (2) chronic articular rheumatism forming adhesions; and (3) tubercle causing early rigidity.

The previous history will indicate whether there has been hæmorrhage into the joint.

Articular rheumatism, producing ankylosis, is as rarely monoarticular as tubercle, producing ankylosis, is polyarticular. Further, the ankylosing form of tubercle is always associated with some thickening of the capsule, and with local elevation of temperature and often with fistulæ, unless the process is completely at an end. Cases of tubercular polyarthritis, wherein the diagnosis remains uncertain for many years, exist, but they are very rare.

The contracture does not always develop into a simple flexion. We often find, especially in tubercle, that the posture is one of slight valgus, with subluxation of the tibia backwards and outwards, as illustrated in fig. 510.

CHAPTER CI.

TUMOURS AND ALLIED STRUCTURES ABOUT THE KNEE-JOINT.

THE knee-joint occasionally presents structures which do not fit in with the previously described bone sarcomata, arising from the femur or tibia, nor with chronic inflammatory processes.

(*a*) Let us begin with the *anterior surface*.

Chronic **prepatellar bursitis** is at once evident, even to the beginner, owing to its superficial position, in front of the patella. The structure may vary in size from an almond to a fist, and accurate anatomical examination shows that it may be just under the skin, under the superficial fascia, or under the deep aponeurosis. Diverticula extending laterally are of importance from the operative standpoint.

If the wall of the structure is strikingly thick and persistently painful on pressure, one should think of **tubercle of the bursa,** which is however, rare. X-rays will show whether it originates in tubercle of the knee-cap.

Occasionally, a change, corresponding to prepatellar bursitis, is found somewhat lower down, situated in front of the patellar ligament, (*Bursitis praetibialis*). Bursitis of the deep *infra-patellar bursa behind* the patellar ligament is still more rare. The swelling has a subdivided appearance, and bulges on both sides of the ligament. The larger it is, the more it interferes with the movements of the joints.

Among the rare tumours of the anterior surface of the joint should be noted, **sarcoma of the patella; lipoma, fibroma** and **sarcoma of the synovial membrane** or the sub-synovial connective tissue. **Lipoma arborescens,** i.e., lipomatous proliferation of the articular fringes, has already been mentioned as occurring in various forms of chronic arthritis and in tubercle. It may be regarded as on the border line of tumour formation. Fibrous polypi which may attain the size of almonds and which feel very much like foreign bodies within the joint, are seen in rare cases of tubercle (Plate IV, fig. *b*). The freedom of the joint movements is characteristic of all these tumours.

(*b*) With few exceptions, swellings in the *popliteal space* are either **extensive bursæ** or **aneurisms.** The distinction is at once evident on palpation and inspection, from the absence or presence of *pulsation.* This, however, may occasionally be absent in recent traumatic aneurisms, and in other aneurisms if their contents are coagulated. But no mistake ought to arise, as the consistence of the structure is comparatively firm, and the patient can always testify to the previous presence of pulsation. Soft or elastic non-pulsatile swellings are, as a rule, enlarged bursæ ; the *bursa under the popliteus*, if situated laterally, and the *semimembranous bursa*, if situated towards the middle line. They may be confused with the very rare lipomata of this region, and also with a cold abscess. Differentiation is easy, if the contents of the swelling can be reduced into the knee-joint, as is often possible in enlarged bursæ. Otherwise, if the swelling is very easily displaced, we should regard it as a lipoma ; if it has an atypical situation, is painful on pressure, and if the movements of the joint are also interfered with, we should regard it as a cold abscess. If the abscess originates in an extra-articular lesion, it may not be possible to diagnose it without a skiagram and an exploratory puncture.

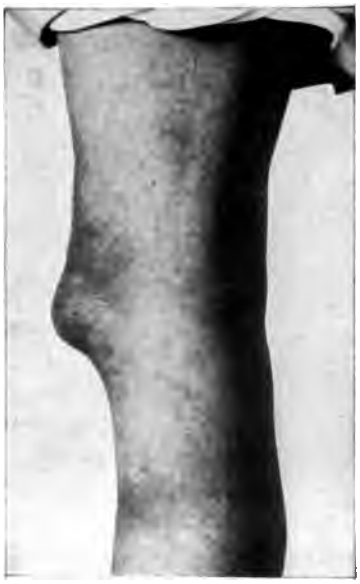


FIG. 511.—Prepatellar bursitis.

Effusion into the bursa may be due to chronic serous (rheumatic) inflammation of the knee-joint, and the bursa may contain loose bodies derived from the joint.

We need only add, in reference to the unmistakable diagnosis of aneurism, that this soon causes neuralgic pains and paræsthesia in the leg, and that it may, after reaching a certain size, fix the joint in semi-flexion. It is due to the same causes as other aneurisms, i.e., trauma, especially stabs and gunshot wounds, arteriosclerosis and syphilis.

CHAPTER CII.

SCIATICA AND OTHER PAINFUL DISEASES OF THE LOWER LIMBS.

MEDICAL nosology contains a number of vague terms which fortunately help to conceal the bitter truth from a patient, without prejudicing the diagnosis, but unfortunately these terms occasionally satisfy the doctor as well as the patient, to the latter's great detriment. Thus a multitude of ills may be embraced under the designations of anæmia, liver trouble, intestinal colic, &c. The same is true of "sciatica." Neuralgic pains of the lower extremity are often summarily diagnosed as sciatica, as if this condition were a clinical entity.

Although references have already been made to this subject, we shall once more describe how a case of "sciatica" should be examined, not only by a surgeon, but by any practitioner.

It is most important to examine the urine and the reflexes—patellar and pupillary—because the condition may be due to diabetes, tabes, or paralysis. Then the surgical possibilities must be thought of. The gluteal region must be palpated, because an obstinate sciatica may be the first symptom of **sarcoma**, originating in the bone or muscle. The course of the nerve must be followed, because sciatica may be due to a malignant growth of the thigh or even lower down. A diffuse thickening of the shaft of the femur, indicating chronic osteomyelitis, may possibly be found. If an injury has preceded the sciatica the question of a foreign body may arise.

A young man fell on a heap of wooden palings, while in an inebriated state. He was subsequently treated for many weeks for "sciatica," and a colleague of mine then succeeded in withdrawing a long piece of paling. The original wound had healed completely over the piece of wood.

If palpation elicits nothing, we must direct our examination towards the spine. Spinal caries, sarcoma of the lumbar vertebræ, or caries of the ileo-sacral joint may simulate a simple sciatica.

If the patient is a young man, of an age when idiopathic sciatica is rare, we should inquire about **gonorrhœa**, and, if necessary, examine the urethra.

I was once consulted by a young man for sciatica. My inquiry regarding gonorrhœa was answered by a decided and resentful negative in the presence of his father. But the youth reappeared on the following day, saying: "I have merely come to tell you that you were quite right."

This cause should also be borne in mind, even in the case of patients of advanced age.

A grey-haired grandmother consulted me for sciatica. She was also suffering from a profuse white discharge. Had her husband not consulted me a fortnight previously for a recent gonorrhœa, I would hardly have diagnosed the cause of her sciatica correctly.

We now proceed to rectal examination. This should never be neglected in a case of sciatica, however objectionable it may be to the patient and unpleasant for the practitioner. We examine in both sexes for cancer of the rectum and for new growth in the pelvis; for malignant disease of the prostate in males and for some disease of the generative organs in the female. In the latter a vaginal examination should also be made. If this systematic examination were invariably practised, we should no longer come across cases wherein women, at the climacteric age, have been treated for weeks or months for sciatica, until at length, an offensive discharge or profuse hæmorrhage has led to a gynæcological examination. The doctor is not always to blame. Female patients often refuse a vaginal or rectal examination because they cannot conceive what bearing it has on their sciatica. The young, inexperienced practitioner may yield to this refusal, but nevertheless he incurs the responsibility for his error of diagnosis. It may be said that such an error is of no consequence to the patient, because a malignant tumour, which has caused sciatica, is already beyond radical removal. Although this may be correct in the majority of instances, it is nevertheless true that an accurate diagnosis is better, not only for the reputation of the practitioner, but also for the interests of the patient and his friends, than futile spa and electric treatment and similar measures. But not all gynæcological diseases which cause sciatica are of malignant nature. Pelvic exudations and incarcerated myomata may irritate the sciatic nerve; indeed rectal constipation may sometimes explain sciatic pains.

Is it possible to distinguish clinically the sciatica which results from malignant growths from idiopathic sciatica? The pain in the former is, on the whole, persistent, in the latter it is rather of a paroxysmal nature. In sciatica due to cancer the patients soon exhibit a peculiar restlessness. Even if the pain is not severe they are still restless, whatever posture they adopt. They cannot be persuaded to sit down, even in the doctor's consulting room; they often persist in walking up and down restlessly. In simple sciatica the disturbances are limited to sensations of pain, there is not usually any loss of sensibility. If, however, it should be present, it does not usually reach any extreme degree. There are never any disturbances of motion. But both these conditions are as a rule present in advanced cases of sciatica, due to compression.

Carcinoma may also produce nerve manifestations, owing to the wandering and proliferation of cancer cells into the lymphatics of the nerve trunk. We must therefore regard "sciatica," which comes on after removal of rectal or uterine cancer, as a recurrence, although we may not be able to detect any cancerous mass compressing the nerve.

If old people complain of severe "sciatica pains" in the leg extending to the toes—pains which often come on quite suddenly and cause limping, we should think of Charcot's "intermittent limp," and should at once inquire whether attacks of pallor or bluish-red discolouration occur in the painful extremity. The occurrence of such attacks indicates the prospect of **gangrene**, especially if on examination the foot is sometimes found to be pale and cold and at other times in a state of venous congestion. This diagnosis would be confirmed by the obliteration of the pulse in the dorsalis pedis artery and posterior tibial, and certainly by the absence of the popliteal pulse. This cause for pain is specially prevalent among old people, as the expression "senile gangrene" indicates. Younger people are, however, not immune from gangrene, but in their case some special cause is responsible, e.g., diabetes, early syphilitic arteriosclerosis, or acute infectious disease, typhoid fever being the most common. I have seen thrombosis of both femoral arteries and eventually of the abdominal aorta, follow a slightly septic finger in a girl aged 20. In some cases, however, no antecedent disease can be discovered. These cases are included under the comprehensive term of Raynaud's disease, the principle indication of which is its symmetry. This condition is attributed to a primary vasomotor disturbance, for want of a better explanation.

The following case belongs to this category, although the affection is unilateral :—

A healthy man, aged 30, who had not suffered from syphilis, began to complain of severe neuralgia of his left foot. There was no obvious cause for this, except perhaps excessive gymnastics. The foot was sometimes pale and cold, at others, bluish-red. Eventually the cyanotic discolouration persisted and gangrene of the foot developed, requiring amputation. Pains and vasomotor disturbances supervened a few years later in the other foot, but gangrene did not follow.

We should think of arteriosclerosis, in the absence of any other cause, if pain is present, even without severe vasomotor disturbances.

Varicose veins should also be included among the causes of pain and "cramp" in the leg, especially in the calf. *Deep* varicose veins are conveniently blamed, when the diagnosis is obscure, because they cannot be seen, and therefore their presence cannot be denied.

On the other hand, sciatica may be mistaken for some other condition—especially the form of sciatica termed *scoliosis ischiadica* (Chapter LXXX). Patients with sciatica often walk with an oblique gait, inclining the trunk, sometimes to the healthy side, at others to the affected side. A beginner, unaware of this habit, may easily devote his chief attention to the scoliosis, and look upon the sciatica as a secondary matter. This view would, however, only be justified in those rare cases wherein the sciatica results from disease of the lumbar spine, such as caries, and the scoliosis is a consequence of the lateral compression of the diseased vertebra. In such cases, all doubt is removed by the tenderness of the affected spinous process and the pain elicited by axial pressure. But in cases of true "scoliosis ischiadica" the condition is one of primary disease of the nerve. The patient endeavours to relax the sciatic nerve by abducting and slightly flexing his leg. Ehret has shown that this posture causes a remarkable approximation of the terminal points of the trunk of the sciatic nerve. The patient involuntarily compensates for the pelvic inclination thus caused by assuming the posture of scoliosis and slight lumbar lordosis.

Recent researches, however, appear to show that scoliosis is more frequently due to involvement of the lumbar nerves in the morbid process.

Experience shows that not all the terminal sensory branches of the sciatic nerve are equally involved. Sometimes the branches to the calf of the leg suffer most, at other times the plantar branches. Stoffel has recently described separate types of sciatica on this basis.

The foregoing remarks regarding sciatica apply *mutatis mutandis* to **neuralgia affecting the anterior crural nerve, the external cutaneous and the obturator nerves.** But as neuralgia of these nerves rarely occurs as an idiopathic affection, it is more likely than sciatic neuralgia to suggest, even to the beginner, some special cause. Search should be made for pelvic tumours, spinal caries, burrowing abscesses, and also for malignant retroperitoneal and inguinal glands. The primary growth may be situated in any part of the area drained by these glands, so that the whole of it will require investigation.

Obturator hernia may be the cause of an obturator neuralgia, but as advice is not usually sought for this hernia until strangulation takes place, it is necessary to inquire for this neuralgia, in order to ascertain anything about it. If it is present, it enables us to exclude internal intestinal obstruction.

Neuralgia of the external femoral cutaneous nerve has been described as a disease, *sui generis*, and provided with the designation of "meralgia paræsthetica." This term should, however, not induce us to abandon attempts to form a more accurate diagnosis. As the position of this nerve exposes it to external damage, we should always think of some isolated or repeated injury (e.g., the friction of an abdominal belt) if there be no other cause of neuritis. It may be mentioned incidentally that this meralgia has been described as a consequence—very indirect—even of flat foot.

Many painful conditions of the foot have received special names, e.g., *talalgia*, *tarsalgia*, Morton's *metatarsalgia*, *pternalgia*, &c. The practitioner is apt to believe that a diagnosis has thus been made, and that there is no further need to search for the cause of the pain. This cause may, however, reside in the most varied conditions of disease. Flat foot is the most frequent of these, but they include the sequelæ of injury, localized inflammatory changes in the bursæ, tendon sheaths, joints, ligaments, fasciæ, and also gout, neuritis (alcohol), and tabes, apart from various neurasthenic pains and the result of badly-fitting boots.

Finally, it must not be forgotten that women often complain of severe pains and paræsthesia in the legs at the menopause. The correct diagnosis is frequently suggested by the fact that similar sensations, although of a less severe character, are experienced in the arm.

CHAPTER CIII.

ULCERS OF THE LEG.

"ULCER of the leg" has become quite a standard type of lesion, so that the beginner is apt to imagine that there is only one variety of ulcer in this region. In addition to the ulcerative processes which occur around the orifices of fistulæ, and which may themselves be of a tubercular nature (fig. 513) if the fistulæ are tubercular, there are three other forms of ulcer which affect the leg, i.e., (1) varicose ulcer, (2) syphilitic ulcer, and (3) cancer of the skin.

Varicose ulcers are usually already diagnosed by the patient. As they preponderate enormously over the other two forms, we are justified in accepting the patient's diagnosis in most cases—not however, before seeing the ulcer. Varicose ulcers vary so much according to the stage in which we see them that we cannot speak of any characteristic appearance. Sometimes we see a brownish-red, hard infiltration of the skin with a circumscribed, superficial, and remarkably painful erosion in its centre. The beginner looks upon this as too trifling to deserve the name of ulcer, until he learns by experience that this erosion, unless it is treated, may develop into an ulcer lasting for weeks and even for months. Sometimes this will occur, even if the erosion does receive treatment.

An ulcer of the leg may present itself as a deeply penetrating loss of substance from the skin, with a necrotic base, and with edges which are serpiginous, steeply shelving, or even undermined. This may be a simple ulcer of the leg without any other supplementary element. The offensive discharge and the inflamed area around merely indicate neglect, either due to social causes or to laziness. At other times we find a flat ulcer with a granulating base and smooth edges, on which new epithelium is developing (fig. 512). Obviously this must be an ulcer on the point of healing, and the duty both of the

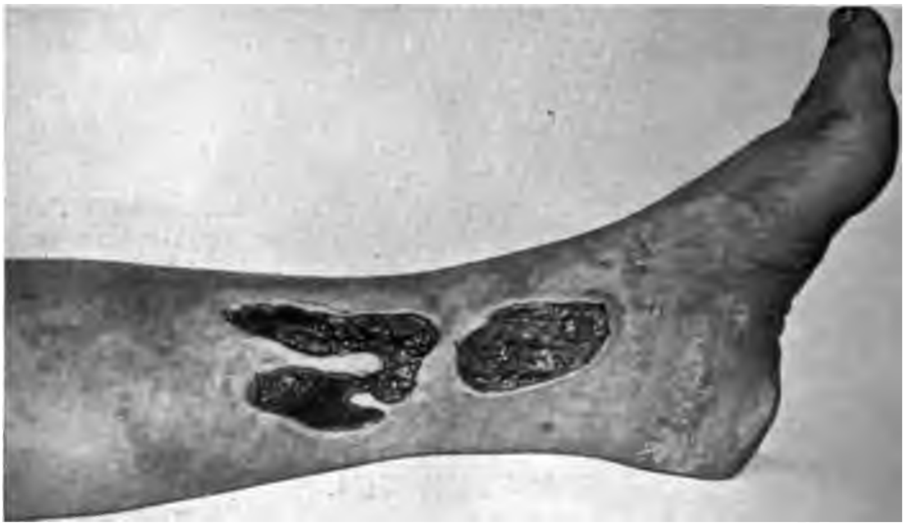


Fig. 512.—Varicose ulcer of the leg.

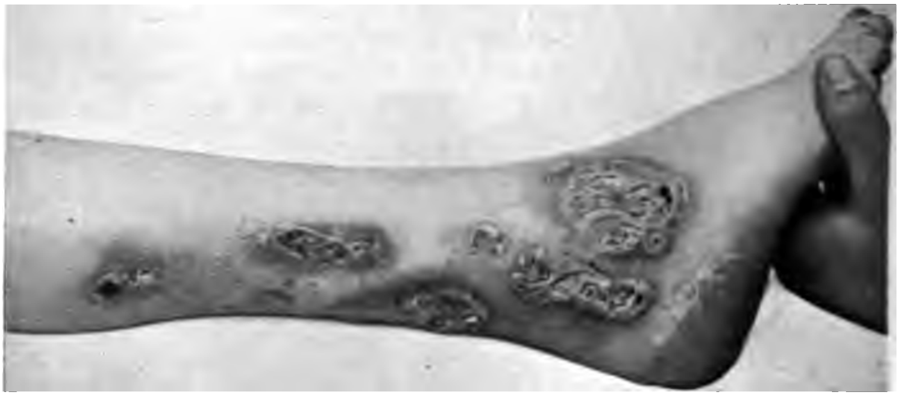


Fig. 513.—Tubercular ulcers of the leg.

patient and practitioner is to do nothing to interfere with the healing process, by unsuitable treatment or inappropriate conditions.

Are varicose veins really indispensable for the diagnosis of a varicose ulcer? As a rule they are present; but sometimes it is necessary to search for them, and for this purpose the patient must stand up for a little while. If the patient has been confined to bed for any length of time, it may be impossible to see even very pronounced varicose veins. The absence of any abnormally dilated veins, does not, however, justify us in assuming a syphilitic basis for the ulcer. It is more likely to have resulted from some indefinite injury, from an abrasion or contusion of the skin, which has developed into an ulcer owing to the unfavourable healing conditions of the leg or through neglect. These ulcers heal rapidly if the patient lies in bed and if the necessary cleanliness is adopted in the treatment of the wound. On the other hand, the presence of varicose veins is not in itself enough to justify the diagnosis of a varicose ulcer. Syphilitic patients may have varicose veins which probably encourage the development of gummatous processes. On the other hand, the so-called "varicose symptom-complex" has often been attributed to syphilitic vascular changes, even if the ulcers themselves do not possess syphilitic characteristics.

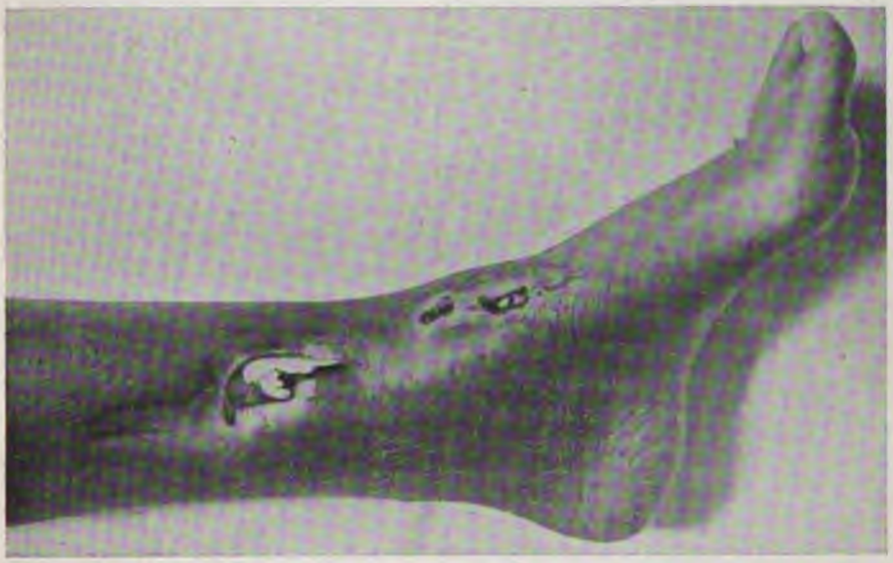


FIG. 514.—Osteomyelitis of tibia, showing scars, sinuses, ulcers, and a sequestrum.

The appearance of the ulcer, as already indicated, shows us the *stage* of the ulcer, but does not of itself tell us anything definitely of its *origin*. Many errors will be made if all ulcers which are somewhat polycircular in shape or have serpiginous edges are put down to syphilis, or if all undermined ulcers are regarded as cancerous. There can, of course, be no doubt about *syphilis*, if the polycircular shape is very pronounced, probably in several ulcers, if there also co-exist round, kidney-shaped or horse-shoe-shaped erosions of the skin, looking as if they had been punched out with a perforating apparatus, and if they had been preceded by well-defined, painless, cutaneous gummata, instead of a diffuse, hard and painful infiltration of the skin. The localization of these morbid changes is very striking in less typical cases. Simple ulcers of the leg are situated in its lower half extending as far as the malleoli. They may affect any part of this region, the front, back, the external or internal surface, and may even become ring-shaped. Ulcers situated higher up towards the knee, or

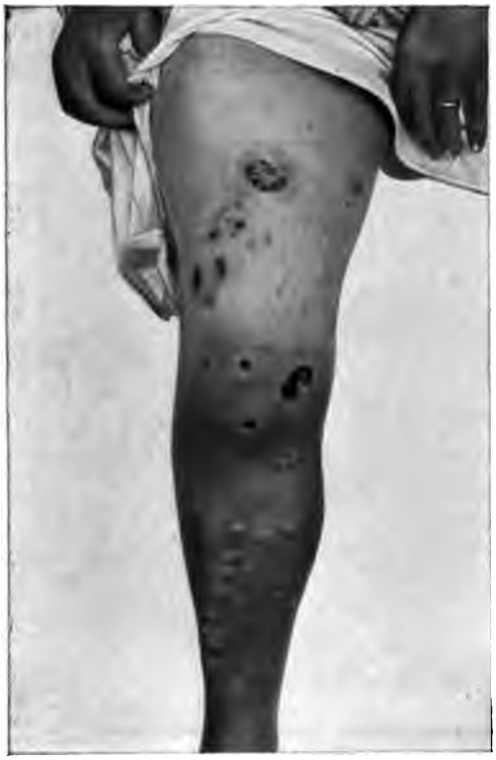


FIG. 515 Tertiary syphilitic ulcers on the right leg, which is also affected by severe varicose veins.

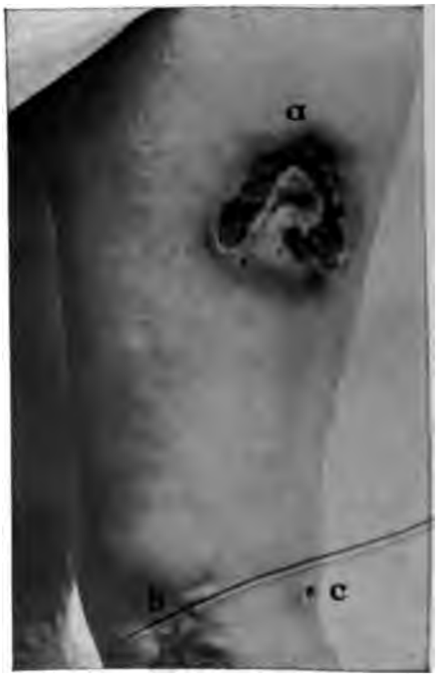


FIG. 516. Same case as fig. 515, one year later. Left leg. *a*, congested horse-shoe shaped ulcer; *b*, an old puckered scar; *c*, cicatrizing ulcer.

lower down on the dorsum of the foot, may indeed be due to injury or to a ruptured varicose vein, but in the absence of such a cause, they are, practically always syphilitic. It occasionally happens that such a syphilide is found directly over the knee-joint, a position in which a varicose ulcer never occurs.

Fig. 515 illustrates the case of a vigorous peasant woman who came to the hospital for "an ulcer of the leg." The diagnosis seemed obvious, and there appeared to be no question of syphilis. She had numerous varicose veins, and had had thirteen confinements without any miscarriage. But the ulcers were on the upper half of the leg, partially in front of the knee, above which was an area of sharply defined red infiltration on the point of softening. There was a puckered scar on the front of the opposite patella, which the patient said was due to a similar ulcer, cured ten years previously by some domestic remedy. This of course settled the diagnosis, and iodide of potassium produced the anticipated result. I learnt subsequently that this patient had been treated twenty years previously for recent syphilis contracted from her first husband. A year later, this patient returned with an ulcer of the left thigh, which is illustrated in fig. 516 and seen to be perfectly characteristic. The puckered scar, just mentioned, is seen at *b* in the figure. The case illustrated in fig. 519, came as one of old-standing "caries" of the foot. There was nothing in the history on which to base the diagnosis of syphilis, but it was suggested partially by the situation and partially by the shape of the ulcer, and also by its yellow fatty-looking base. This diagnosis was confirmed by the result of treatment.

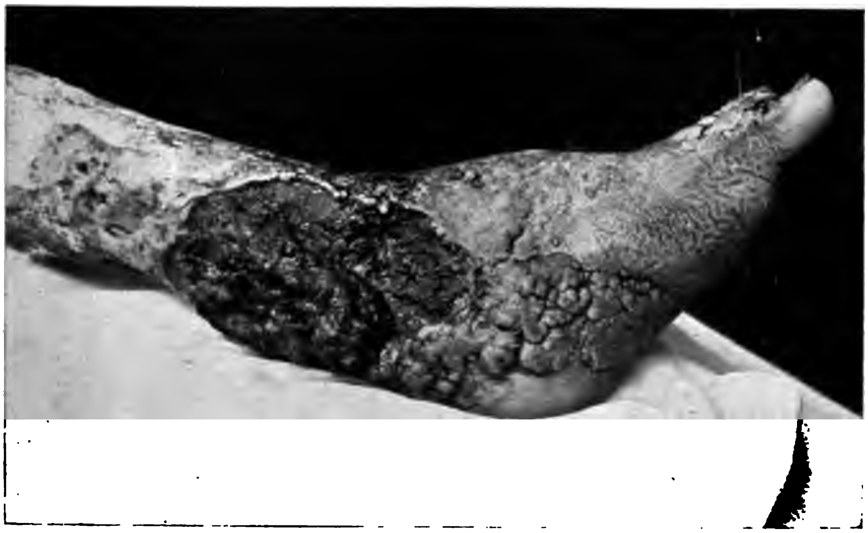


FIG. 517.--Cancerous degeneration of ulcer of the foot.

It has been stated above that undermined edges are not enough to arouse the suspicion of *cancer*. Indeed, an ulcer without undermined edges may be cancerous—although ordinarily this condition is an important sign. If an old scar breaks down and ulcerates—old scars have a decided tendency to become cancerous—and not only refuses to heal, but actually increases in extent, this indicates a tendency to

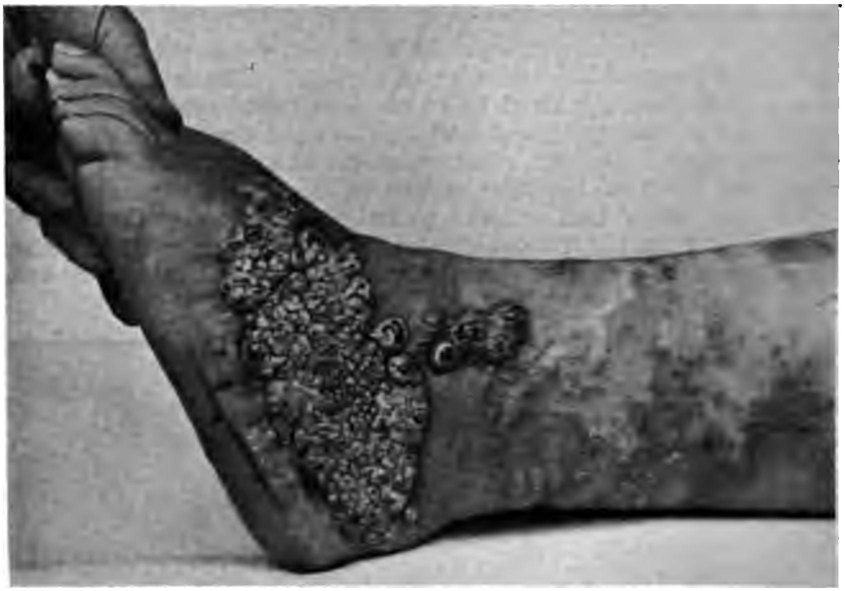


FIG. 518.—Scars of syphilitic ulcers with papillomatous hypertrophy of the skin of foot.

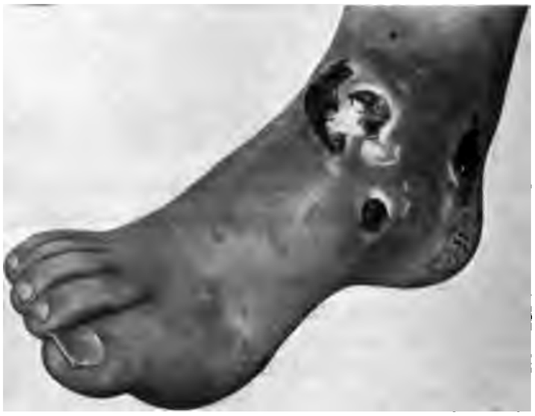


FIG. 519.—Tertiary syphilitic ulcer of the foot.

cancer. If the ulcer does not become covered with healthy red granulations, this constitutes a more important sign, and we must entertain some doubt. If the base constantly remains granular in appearance and if the well-known little whitish plugs can be squeezed out of the more recent portions, a histological examination of a piece of the margin is demanded forthwith.

Sometimes the diagnosis of cancer is not made until the onset of enlarged glands in the popliteal space and groin. This occurred in the case of a young man, whom I saw while acting as an assistant. A very obstinate ulcer developed on an old scar due to a burn. The youth of the patient disarmed any suspicion of cancer at first. But the onset of enlarged glands led to a histological examination and a diagnosis of cancer was made.

A papillomatus hypertrophy of the skin of the foot often occurs as an expression of the chronic circulatory disturbance, associated with ulcers of the leg (figs. 518 and 519). This condition must not be mistaken for cancer. It is really connected with the diffuse elephantiasis which is often observed under similar conditions.

Lupus of the leg, independent of any bone lesion, is rare, and therefore its differential diagnosis is of slight importance. It is recognized by the same features as in other situations. In cases of doubt a histological examination is necessary.

CHAPTER CIV.

SWELLINGS AND TUMOURS OF THE LEG.

SWELLINGS and tumours of the leg present similar conditions to those which we have considered in detail in connection with the thigh. But there are certain features peculiar to the leg. We will begin with—

I. *Diseases of the soft tissues.* Fibromata and fibro-neuromata (fig. 525) deserve first mention among the acute swellings. The principal swelling, not of the nature of a new growth, is that of Elephantiasis, of varied origin. In the absence of tropical *filarial disease*, it is usually due to some congenital vascular anomaly, but it may also be one of the indications of the so-called **varicose symptom-complex.** The **varicose veins** constitute the chief symptom of this latter, and a few remarks about them are necessary.

There can be no difficulty in diagnosis if the serpentine course of the veins is visible, and if their lumen is clear, so that slight pressure or a change of posture suffices to empty them. Diagnostic interest centres around the questions whether the veins belong to the large or small saphenous group and whether the valves in the former have become incompetent. Trendelenburg's sign is useful in this connection.

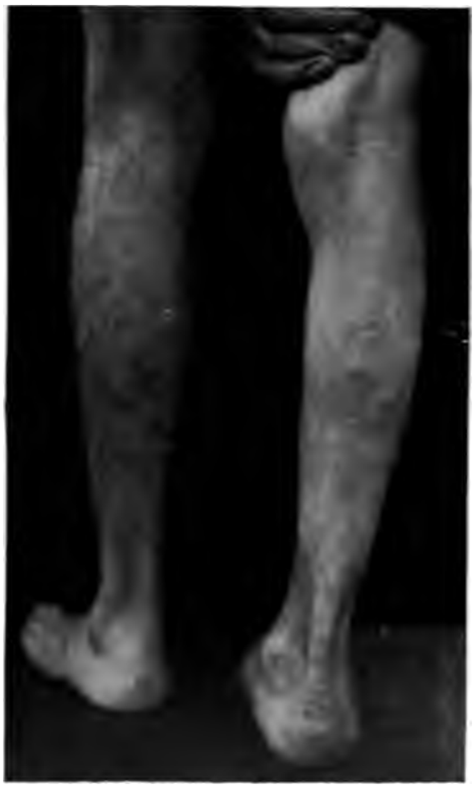


FIG. 520*a*.—Varicose veins, after the limb has been emptied of blood and the root of the large saphenous vein is being compressed.

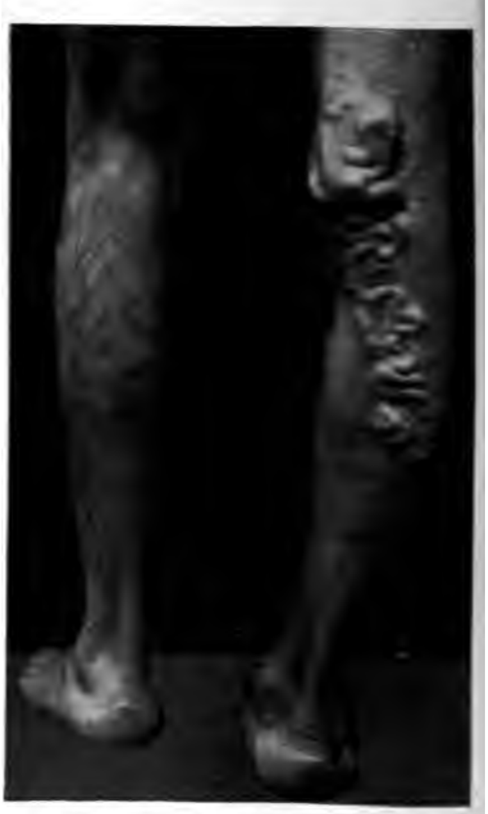


FIG. 520*b*.—Same case, after relaxation of pressure from the vein (Trendelenburg's sign).

The patient lies down, to empty the veins of the limb. The root of the large saphenous vein is then compressed with the finger and the patient is instructed to stand up. If the veins remain empty but fill up as soon as the pressure is relaxed, it is obvious that the large saphenous vein is affected (*cf.* figs. 520*a* and *b*).

Even if the veins are thrombosed, the diagnosis is easy, as long as separate serpentine cords are recognizable. The beginner may be uncertain when confronted by an isolated thrombosed convolution of veins, looking like a tumour, but even then the history will give a clue, and the patient will probably have made the diagnosis already.

In certain rare cases, Elephantiasis is associated with congenital defects in the development of the lymphatics and blood-vessels. It may then be merely one indication of a neuro-fibromatosis, which is either generalized, or localized to one limb (fig. 522).

II. *Changes in the bone* are for anatomical reasons more accessible to examination than in the case of the thigh, and their diagnosis is therefore easier. We begin with acute changes.

A.—ACUTE DISEASES.

Acute osteomyelitis constitutes the type of the acute diseases. The patient is usually an adolescent, who is suddenly seized with severe feverish symptoms, and a more or less extensive intensely painful swelling over the tibia, which rapidly becomes œdematous and ruddy, culminating in fluctuation. There can be no hesitation about the diagnosis, if one bears in mind the pathological diagram of the disease illustrated in fig. 529, and co-relates it with the various stages of the disease. In certain cases the diagnosis is facilitated by two circumstances, viz., an injury a few days before the onset of the illness, or the presence of a suppurating lesion (e.g:, furuncle) somewhere in the body.

We have already referred to the fact that the neighbouring joints are often involved in the inflammatory process. This is of diagnostic importance, because the original bone lesion may be overlooked owing to the bone disease.

The clinical condition can only give a clue to the bacteriological diagnosis, if it has been preceded by a specific infection, e.g., a furuncle (*Staphylococcus aureus*), typhoid fever (typhoid bacillus), pneumonia (Fränkel's diplococcus). If there be no indication of this kind we may assume that the disease is staphylococcal in origin.

Inflammation limited to the soft tissues or periosteum is sometimes mistaken for osteomyelitis. This also applies to the transient swelling of the soft tissues which are occasionally seen in endocarditis (with streptococcal infection of the blood). An example of this has been quoted in connection with sepsis due to ear disease.

B.—CHRONIC INFLAMMATORY PROCESSES AND TUMOURS.

The difficulties we have encountered in making a differential diagnosis between chronic inflammatory processes and tumours in the thigh are magnified in connection with the leg, because syphilis and tubercle play a part in this region which they do not in the thigh.

The following case will indicate the difficulty of diagnosis in certain circumstances. A man aged 68 had noticed a swelling of a year's duration on the superficial surface of the tibia, and as it was painful, ascribed it to rheumatism. On medical advice a skiagram was taken, but this was inconclusive. The patient then escaped medical supervision, and the swelling gradually increased. Its localization and consistence suggested a gumma, especially as the patient had suffered from syphilis. The Wassermann reaction was negative, but this was no definite contra-indication. The painful character of the disease also suggested some acute inflammatory condition, which could have been an osteomyelitis due to the typhoid bacillus or to the staphylococcus, as the patient had suffered from typhoid fever, and had recently been the victim of a furuncle. The agglutination test for typhoid was negative. Another skiagram showed more loss of bone than new formation, a point in favour of

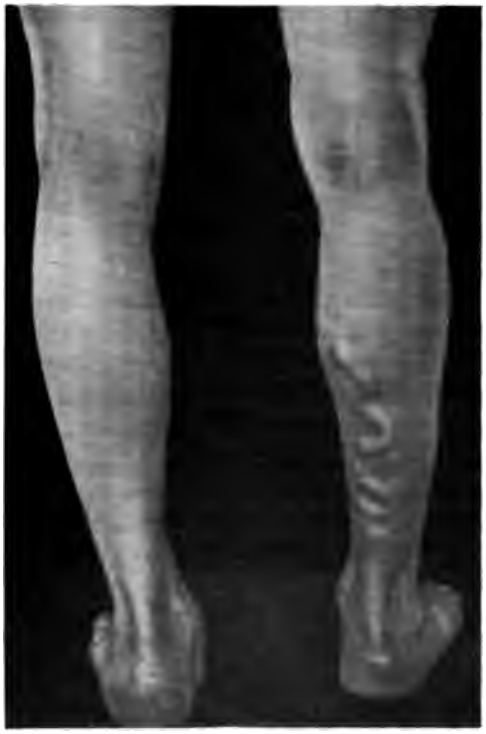


Fig. 521. Varicosity of lesser saphenous vein.

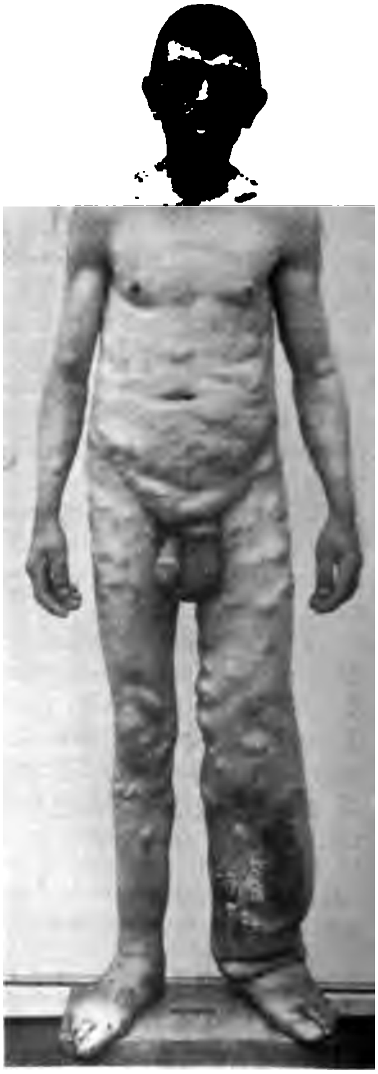


Fig. 522. — Neuro-fibromatosis with elephantiasis of left side.

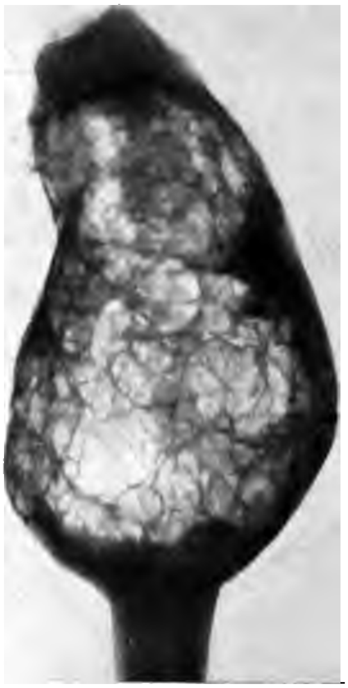


FIG. 523.—So-called aneurism of the fibula. Skiagram of the specimen obtained by operation. (Cystic disease of bone).

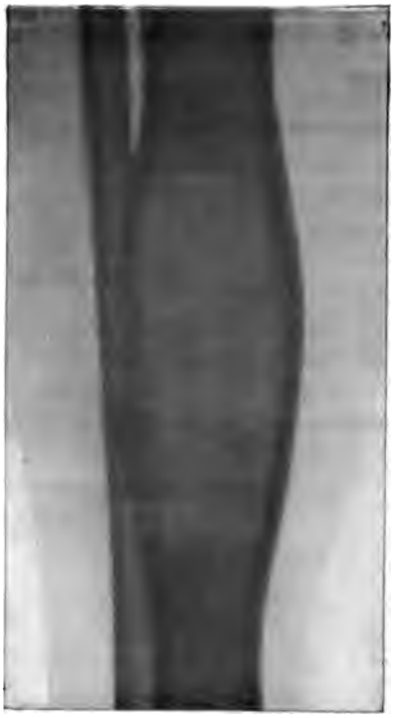


FIG. 524.—Fibrosarcoma of tibia originating in the medulla.

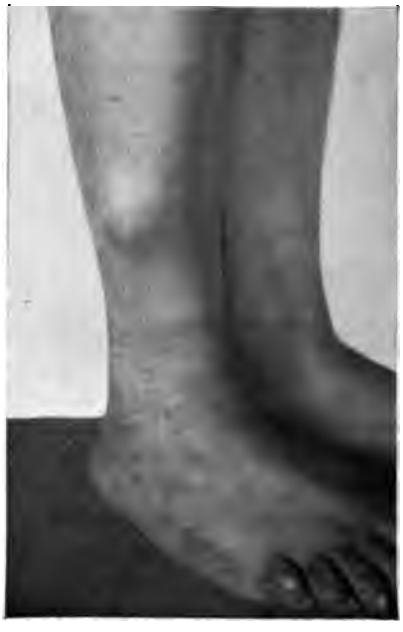


FIG. 525. Neurofibroma of the superficial peroneal nerve.

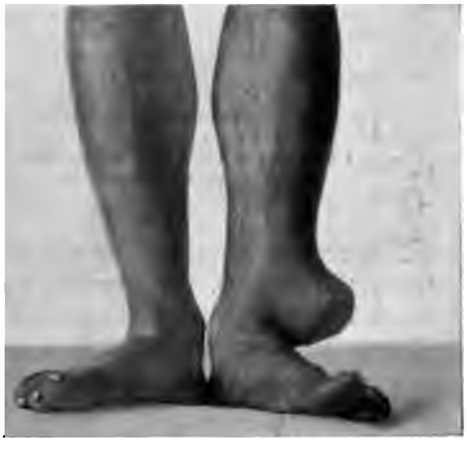


FIG. 526.—Chondroma of the tibia.

tumour. A positive diagnosis could only be made by an exploratory incision and this revealed a *sarcoma*.

In order to obtain a bird's eye view of the subject, we should distinguish (1) Diffuse inflammatory processes, and (2) circumscribed swellings.

(1) Diffuse Inflammatory Processes.

Diffuse swellings should suggest a mild form of **osteomyelitis**, or in contrast with what we have seen in regard to the femur.

Syphilis.—If the disease is *acquired*, the diagnosis of gumma is established by the presence of isolated, circumscribed, and scattered inflammatory areas on the anterior surface of the tibia quite apart from the history. In chronic osteomyelitis, the thickening of the bone is usually more diffuse, which is again a contrast to gumma (fig. 530). Localized abscesses, which heal up after the extrusion of a small sequestrum, often occur in this condition. The problem is, however, quite different in the case of children. *Hereditary syphilis* of the tibia is not usually of a gummatous nature, but is

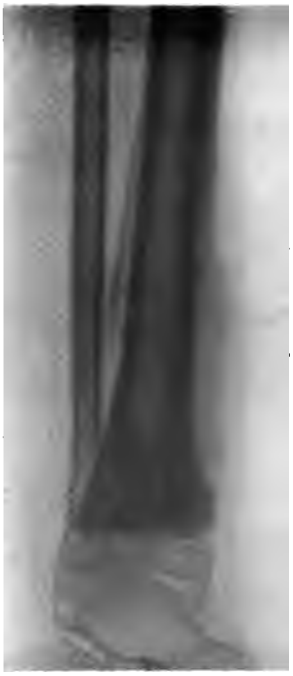


FIG. 527.—Sarcoma of tibia, with radiating bone proliferation.

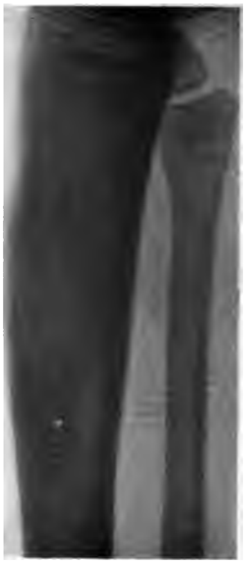
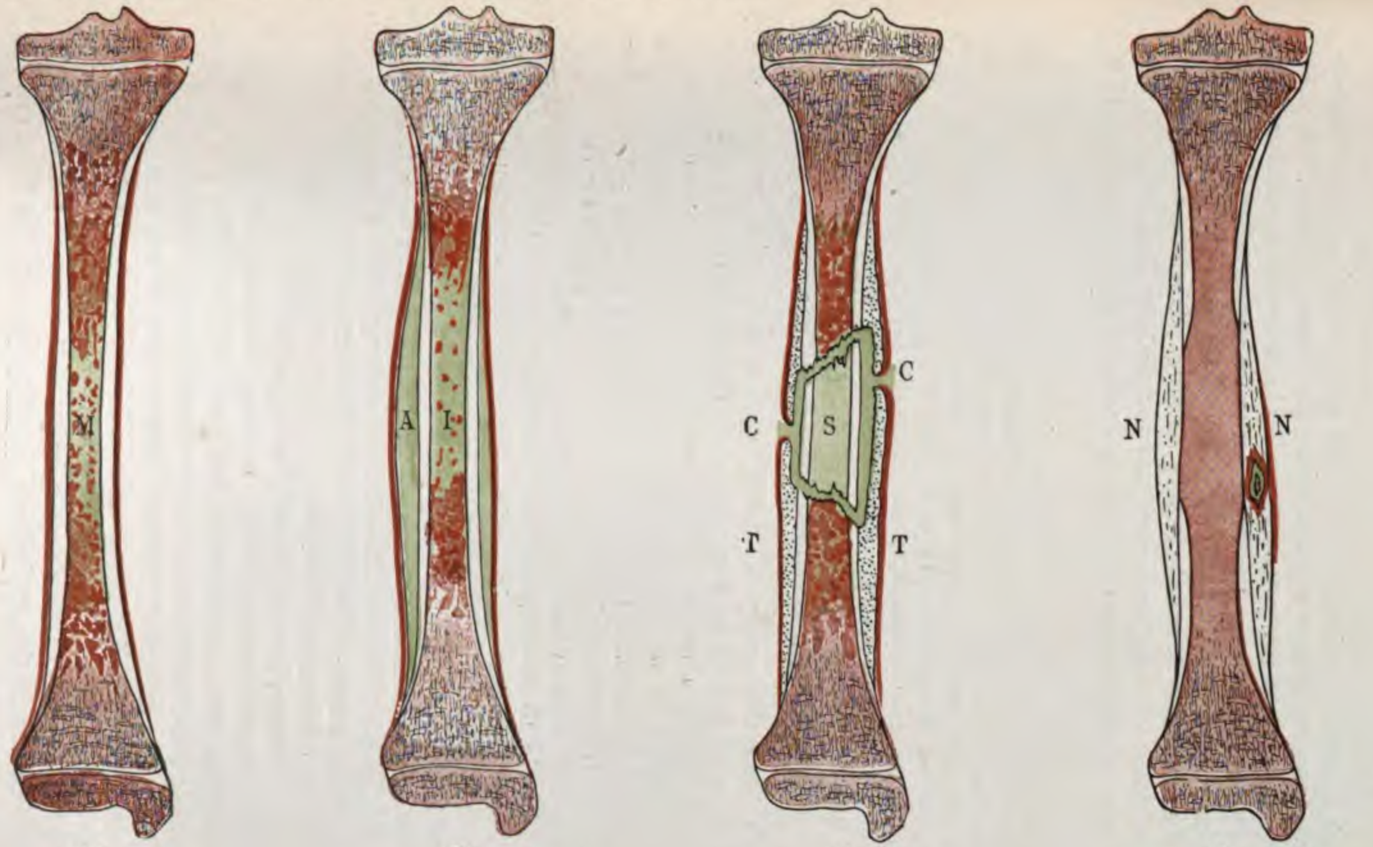


FIG. 528.—Congenital syphilis of tibia. Circumscribed form.

Bright red = normal bone marrow. Red = inflammatory exudation. Green = pus formation.



(*a*)
Inflammatory infiltration of the bone marrow. M = Early suppuration therein. Inflammation of the periosteum.

(*b*)
Suppuration within (I) and without (A) the bone. Periosteum stripped up by the pus.

(*c*)
Formation of ring sequestrum (S) Periosteal new bone formation. T = sheath of new bone formed in necrosis, with C cloacæ. Granulation formation in medulla of diaphysis.

(*d*)
Sequestrum replaced by newly formed bone (N) of compact character. Regeneration of the bone marrow. Secondary formation of a small cortical sequestrum in the newly formed bone.

FIG. 529.—Course of acute osteomyelitis of tibia.

recognized by diffuse infiltration of the periosteum, and in its subsequent course by diffuse thickening of the bone (fig. 531). Syphilis often proceeds just like osteomyelitis, by means of exacerbations, but it is differentiated from the latter by the absence of elevations in temperature and by its symmetry. The tendency of the tibia to bend with the convexity forwards is very striking. Obviously the history is conclusive. It may be mentioned incidentally that the tibia and the bones of the forearm are the favourite sites for these lesions of congenital syphilis.

A girl, aged 7, suffered from periodical attacks of painful swelling of both tibiæ. A diffuse tender thickening of the bone could be demonstrated (see fig. 531). There was no pyrexia. The previous treatment had been directed against tubercular disease. The only indication of a syphilitic heredity was the mother's statement that the father occasionally suffered from an ulcerative skin lesion. Antisyphilitic treatment caused the symptoms to disappear in a very short time.

(2) Circumscribed Swellings.

We limit this group to those cases wherein the swelling and tenderness do not extend beyond one half of tibia, but which definitely originate within a much smaller compass. The principal diseases which enter into consideration are (1) the isolated **gumma**, (2) the circumscribed chronic forms of **osteomyelitis** (usually staphylomycosis), (3) **tubercle**, and (4) **sarcoma**.

We have already dealt with syphilis. In regard to osteomyelitis and tubercle, the most prominent objective signs are the swelling and new bone formation in the neighbourhood of the periosteum, so that the inexperienced observer is liable to be content with the diagnosis of *periostitis*. But the more carefully these cases are examined and the more often the assistance of a skiagram is invoked, the more frequently will we find that this periostitis is due to changes in the bone marrow, either of the nature of an abscess or of an area of granulation, with or without sequestrum formation.

Is it, however, possible to tell, by inspection whether the disease is tuberculosis or osteomyelitis? It must be confessed that clinical examination often leaves us completely in the lurch. We will consider separately the diseases of the diaphysis and the epiphysis:—

(*a*) It was previously assumed that disease in the *diaphysis* could only be tubercular if it occurred in children; disease in the diaphysis in adults was always considered to be osteomyelitis. But as bacteriological examination became more frequent, it was seen that tubercular disease occurs in the medulla of the diaphysis of the tibia, even in adults. To a considerable extent, we must depend upon the history for the distinction. An acute feverish onset with exacerbations of the same character denote osteomyelitis, but these symptoms are not always present. A gradual onset and a gradual increase of symptoms denote tubercle, but this may also take a sudden turn for the worse. The diagnosis is easier if the disease follows an acute infection such as typhoid fever, scarlet fever, &c. We shall probably be correct in attributing the cause to the organism of the primary disease (e.g., typhoid bacillus), or to secondary infection by one of the ordinary pus organisms. The circumscribed osteomyelitis illustrated in fig. 532 followed a case of whooping-cough.

If no conclusion can be arrived at, either from the history or from the rest of the physical condition—other tubercular or other osteomyelitic foci—we must be content with the anatomical diagnosis, an abscess of the bone. The tuberculin reaction may sometimes throw light on the case.

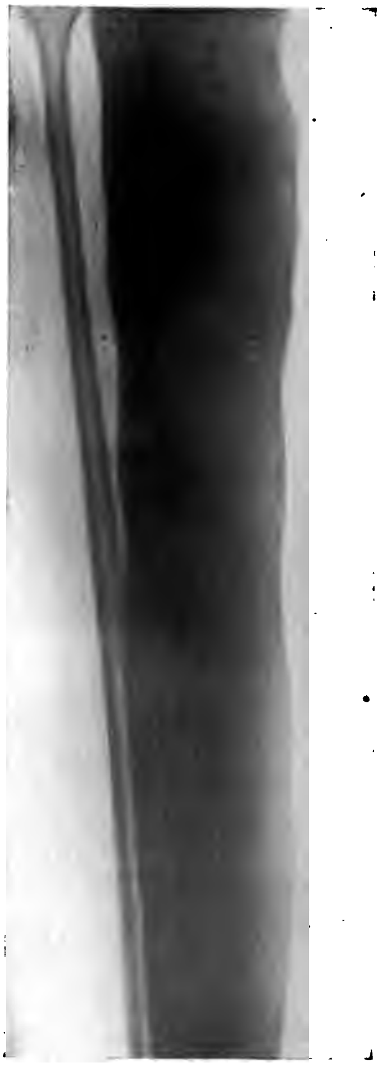


FIG. 530.—Old diffuse osteomyelitis of the tibia.

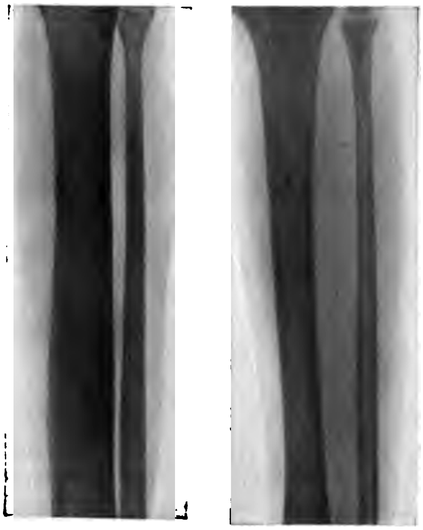


FIG. 531.

(*a*) Periostitis of the tibia due to congenital syphilis.

(*b*) Normal tibia in child of the same age.

If the abscess is situated immediately under the skin, cultures should be made from the pus obtained from an exploratory puncture, and within two days it can be ascertained whether organisms of acute suppuration are present or not. If they are not present the case is probably tubercular. If pus cannot be obtained without operation, the bacteriological examination must be made supplemented by animal inoculation, if necessary, after the operation.

A precise diagnosis is requisite, both for prognosis and treatment.

It is generally recognized that a lesion in the epiphysis is significant of tubercle. If the clinical picture does not however quite resemble an inflammatory process, and if the shape of the swelling is irregularly nodular, or if its size exceeds that of an ordinary circumscribed inflammation, we should think of a new growth. This is most likely to be a sarcoma, or if it developed very gradually, a fibrosarcoma. Pure fibromata of bone are very rare. The diagnosis of chondroma, osteoma and osteochondroma is simple, whether they are strictly limited in size, or project from the bone in grotesque shapes (fig. 526). Derangements in the adjoining nerves in the form of severe neuralgia, anæsthesia, or even paralysis support the diagnosis

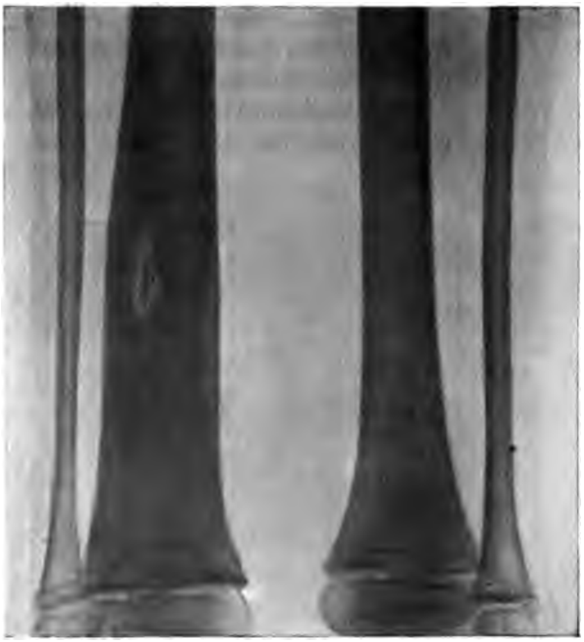


FIG. 532.—Localized subacute osteitis after whooping-cough. A small sequestrum is seen in an abscess surrounded by a sclerosed area.

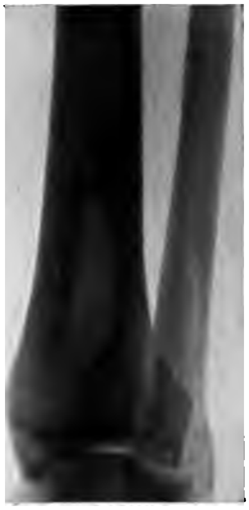


FIG. 533.—Chronic abscess of bone in the lower end of the tibia.

of sarcoma. These are, however, somewhat late symptoms, and our diagnosis ought to be early.

If the tumour cause a spindle or beet-root shaped distension of the bone; if on examining a parchment-like crackling is detected, and if a vascular murmur is heard on auscultation, we may assume the presence of bone cyst full of blood. This has been termed *ostitis fibrosa cystica*, or *aneurism of bone*. Some attribute it to an antecedent injury, others regard it as a comparatively innocent sarcoma, with central degeneration. Fig. 523 is a good illustration of the condition.

A skiagram is generally appealed to, in order to clench the diagnosis between inflammation and new growth; but we may even then be left in the lurch. A central sarcoma is not always distinguishable from a central abscess. If there is pronounced periosteal thickening, it points with great probability to an abscess, but slight periosteal deposits may also occur in sarcoma, although they are usually considered to be due to inflammatory processes. If bony tissue penetrates the pathological structure in radiating lines, it is very significant of sarcoma (fig. 527).

The X-ray picture of osteo-chondroma yields a blotted skiagram, which facilitates the diagnosis.

Sarcoma must be thought of, in connection with swellings of the epiphysis as well as of the diaphysis. We have already mentioned the points which should turn our thoughts in this direction.

I once saw a case of sarcoma of the head of the tibia in a girl aged 20, with symptoms so acute as to suggest osteomyelitis. The diagnosis was only made by histological examination, after an exploratory incision.

The main feature by which a sarcoma is recognized in a skiagram is a hazy transparency in the spongy tissue. In osteomyelitis the periosteal changes predominate; and it is almost always possible, after a time, to demonstrate an osteosclerotic zone around the centre of the infection, both in osteomyelitic and in tubercular lesions of the epiphysis.

The remarks concerning primary sarcoma hold good for secondary growths in the tibia, which occur chiefly after cancer of the breast, malignant goitre and hypernephroma.

CHAPTER CV.

INJURIES ABOUT THE ANKLE-JOINT.

In examining an ankle after an injury, the most important practical consideration is to ascertain whether there is any deformity or not. Indeed we shall adopt the absence or presence of deformity as a basis of classification, although at first sight it may not appear to be very scientific.

(1) INJURIES WITHOUT DEFORMITY.

If the shape of the foot remains normal after an injury, or at any rate is only slightly swollen, we may forthwith exclude dislocation and fracture with displacement. This at once limits the diagnosis to contusions, sprains, or fractures without displacement.

The diagnosis of **contusion** requires no detailed consideration, because it is easily inferred from the nature of the injury and presents no difficulty whatsoever.

The diagnosis of **sprain** is made by exclusion, just as in the wrist and elbow; i.e., it can only be entertained if it is quite certain that there is no **fracture** present. The *history* is, however, often very suggestive. We may diagnose a sprain, if the patient, after sustaining an injury—not necessarily a severe one—to his ankle, complains of gradually increasing pain and tension, which do not entirely disappear, even on complete rest. On the other hand, a severe pain at the moment of the injury, which subsides when the limb is kept at rest, and which returns when any movement is attempted, would *a priori* suggest a fracture.

The explanation is quite simple. Pain which is not severe at the moment of the injury, but which gradually increases and does not disappear in spite of rest, indicates an effusion of blood within the joint—a circumstance which occurs in a sprain. A fracture may also lead to intra-articular effusion, but as the fracture opens up a path for the blood in the surrounding tissue, the effusion is under less tension. The general experience of fracture is, that the pain disappears when perfect rest is maintained once the fracture has occurred, and that it only returns as a result of movement. But it must not be assumed that all sprains present the same clinical features, for in some, laceration of the extra-capsular structures is the predominant lesion, and the pain therefore occurs mainly on movement, just as in fractures.

Loss of power of movement is by no means decisive. The beginner is apt to diagnose a sprain if the patient is able to walk, and to diagnose a fracture if he is not able to do so. But, as a matter of fact, the position may be quite reversed. We often see that a patient who has a subperiosteal fracture of the malleolus—especially the fibula—is able to walk, whereas a patient with a severe articular effusion will anxiously avoid the least step, even if the bone is not injured.

The *localization of the pain on pressure* is of great importance for diagnosis. But before proceeding to palpation, we should test the *pain produced by axial pressure.* If this pain is pronounced, it indicates fracture of the tibia above the malleoli, or fracture of the tarsus. There is usually no pain on axial pressure in simple malleolar fractures without displacement. We now palpate the joint. A diffuse tender swelling over the entire anterior surface indicates an effusion of blood under tension—most probably a sprain. We next examine the ends of the bones of the leg. If the tibia presents a narrowly circumscribed area of tenderness above the joint line, traversing the entire thickness of the bone, it is quite certain that a supramalleolar fracture exists, or, in a young person, a separation of the epiphysis. The same condition will usually be found in the fibula, either higher up or lower down. If the continuity of the tibia is unbroken we should palpate the internal malleolus. Circumscribed tenderness of its extremity indicates that the internal lateral ligament is torn off or lacerated, thus constituting a sprain. If the tenderness runs transversely over the

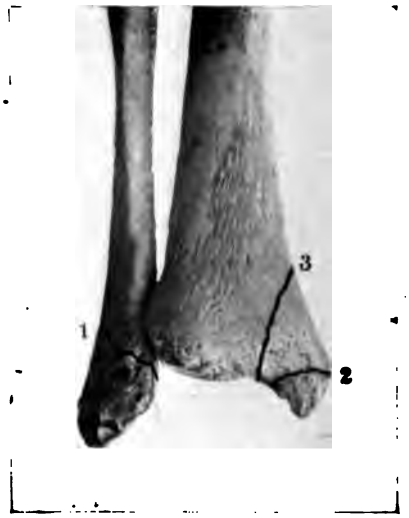


FIG. 534.—Fractures of malleoli through adduction. Fracture lines 2 or 3 may occur alone; fracture line 1 occurs in combination with 2 or 3.

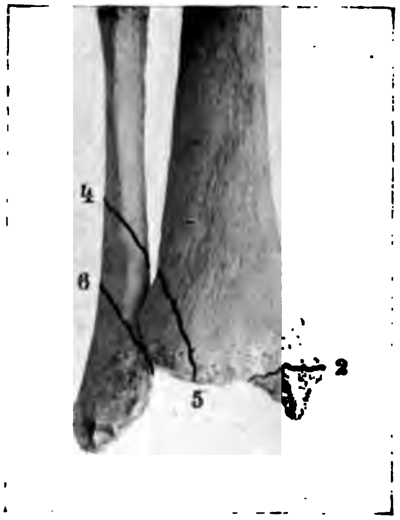


FIG. 534.—Fractures of molleoli through abduction. Fracture lines 2, 4 or 6 may occur alone. Fracture line 2 may occur in combination with 4 or 6; or 2 may occur with 4 and 5.

malleolus (fig. 537), or obliquely or even directly upwards (fig. 536), we must assume that a fracture exists, although it may be impossible to feel any fissure, sharp edges or false mobility. We examine the external malleolus in a similar manner. Tenderness at its extremity indicates laceration of a ligament; tenderness higher up points to a fracture. False mobility can be obtained much more frequently than

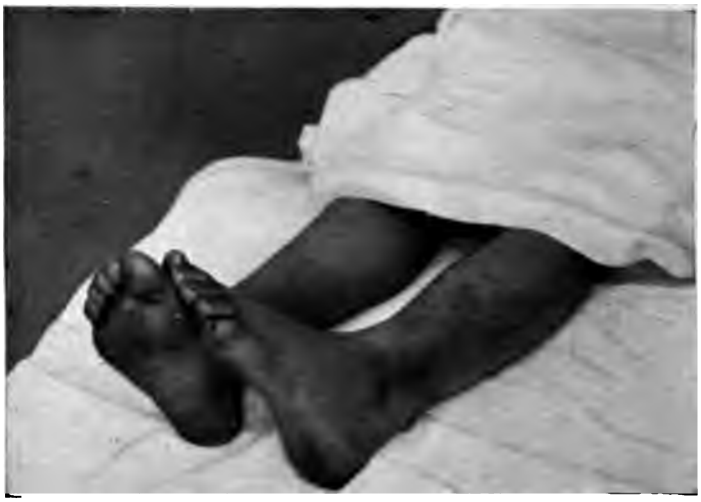


FIG. 535.—Adduction fracture of left internal malleolus.

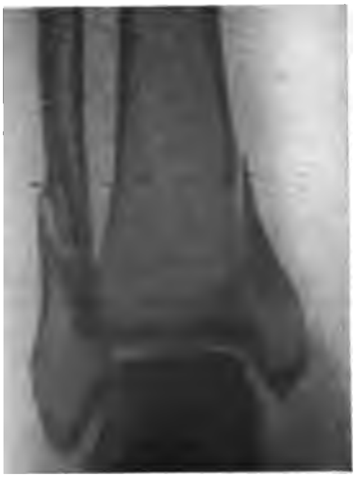


FIG. 536.—Adduction fracture of internal malleolus, with torsion fracture of fibula.

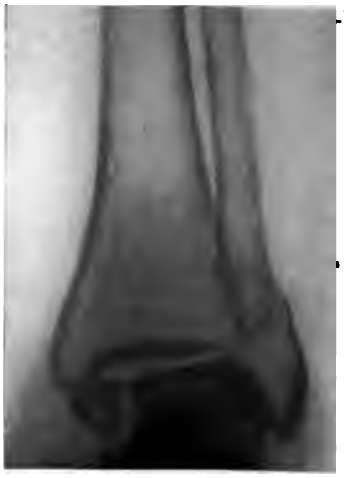


FIG. 537.—Deep seated fracture of external malleolus and fracture of internal malleolus. (Abduction fracture.)

on the inner side, because we can move the lower fragment with the tip of the index finger, but it cannot always be elicited.

If there is distinctly localized pain on pressure, apart from the tips of the malleoli, it is almost certainly indicative of damage to the bone. There are cases, however, wherein this localized pain appears to be due to a detachment of a ligament or piece of periosteum, extending high up. It is obviously necessary to take into consideration the precise type of pain, which is present.

Indirect pain on pressure is another, though less constant sign, which points to fracture of the fibula. This sign is obtained by pressing the fibula against the tibia in the middle of the leg. If the patient then feels a circumscribed pain lower down, there can be no doubt about the presence of a fracture, or at any rate of a fissure. In supramalleolar fracture of the tibia, the fibular fracture is often at its upper end and is easily overlooked.

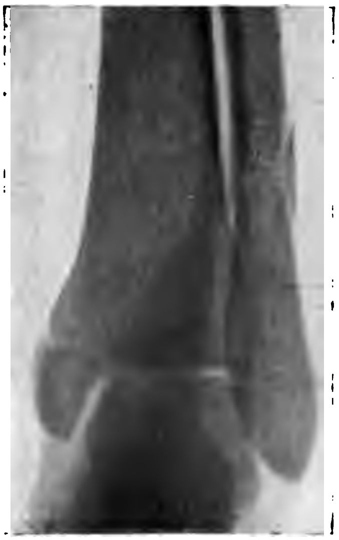


FIG. 538. — Abduction fracture with cracking of fibula, in classical position.

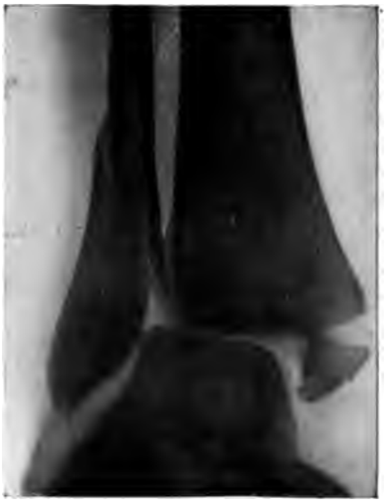


FIG. 539.—Torsion fracture. Detachment of external malleolus.

If we are not in a position to confirm our diagnosis by means of a skiagram, confirmation will be derived from the angular shaped ecchymoses which form below and behind the broken malleolus, in the course of two to three days. Similar ecchymoses may, however, also appear after severe sprains.

It is of some interest, from the points of view of treatment, to recognize the *mode of origin of the fracture* and the ***position and*** *direction* of the line of fracture. Fractures of the malleolus are divided into those produced by violent adduction, by abduction and by eversion of the foot.

In an *adduction fracture* the internal malleolus is always broken off, either in the same plane as the joint line, or in an oblique or vertically ascending direction. A fracture of the external malleolus is often associated with it at about the same level as the joint cleft. The fracture in the former instance is produced by bending, and in the latter by tearing.

In an *abduction fracture* the internal malleolus is torn off near its base; skiagrams show that the fibula is broken at what is considered to be its weakest spot, i.e., about 5 or 6 cm. above the tip of the malleolus. It is, however, frequently cracked much lower down (fig. 537). There is often, in addition, a detachment or a fracture of a wedge-shaped fragment of the outer margin of the tibia (fig. 534). In *fracture produced by torsion* the signs are similar to an abduction fracture, i.e., supramalleolar fracture of the fibula, sometimes a wedge-shaped detachment of the external margin of the tibia and generally a detachment of the internal malleolus. The fracture of the fibula does not, however, present the appearance of a fracture produced by cracking, but it is of a spiral shape (fig. 539).

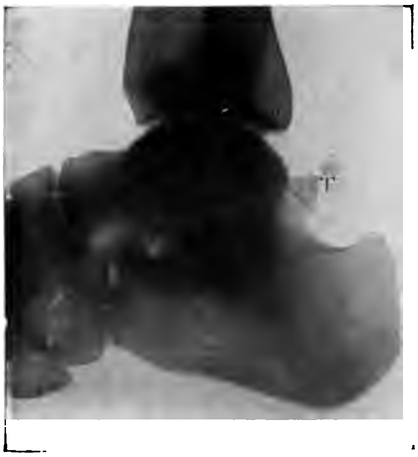


FIG. 540.—Os trigonum (T).

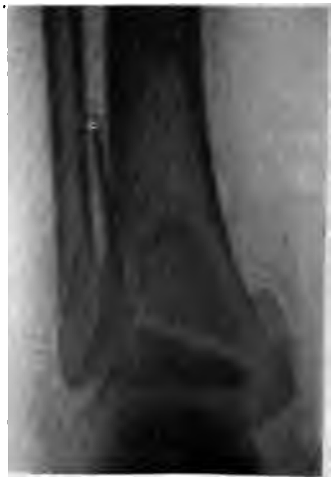


FIG. 541.—Abduction fracture of both malleoli, with dislocation of foot outwards.

It is not safe to base the diagnosis exclusively on the position of the foot. The position of adduction (fig. 535) is fairly conclusive of an adduction fracture, but abduction may be the secondary result of attempts at walking, even if the original injury has been an adduction fracture.

Over excessive *dorsal or plantar flexion* are rare causes of fracture; the former leads to the detachment of a piece of the anterior surface of the tibia (Lauenstein); the latter leads to a detachment from the posterior edge of the tibia, or its posterior surface. This latter fracture was termed by French surgeons, long before the Röntgen period, "fracture marginale postérieure du tibia," and it has lately been described, with the aid of skiagrams, by Meissner of von Brun's clinic. The fragments in both cases are usually triangular in form. The diagnosis is based upon the etiology, the results of palpation and the skiagram (figs. 543 and 545). Corresponding fractures may also be produced by the laceration involved in the reversed movements. Leuenberger has proved from the material in our clinic, that the detachment of a fragment from the posterior surface of the tibia is an injury which occurs at about the same age-period as the separation of the epiphyses, the epiphyseal line still partially persisting, i.e., from the thirteenth to the fifteenth year (fig. 542).

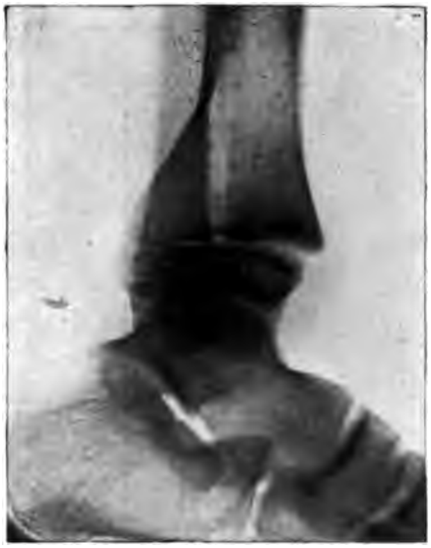


FIG. 542.—Separation of the epiphysis, with detached bone from the posterior surface in a lad aged 15.

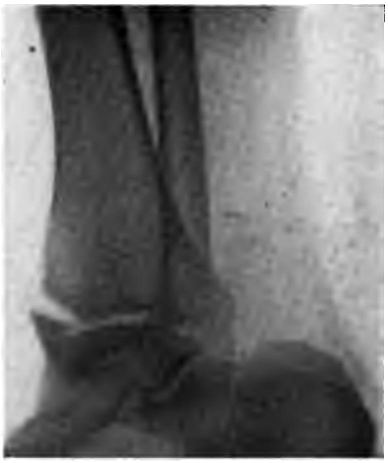


FIG. 543.—Detachment of bone from anterior margin of tibia. Fracture of fibula.

If no lesion can be found in connection with the malleolus, but the patient nevertheless complains of sharp pain as soon as he puts his weight on the foot, so that walking is impossible, we must palpate the tarsal bones, especially the astragalus, as far as this is accessible. An effusion of blood on the anterior surface of the ankle, and great tenderness over the head of the astragalus with severe pain on dorsiflexion, render it very probable that a **fracture of the astragalus** has occurred.

A positive diagnosis cannot be made without a skiagram. In interpreting it, however, it should be remembered that a small Wormian bone (os trigonum) is occasionally seen at the posterior end of the astragalus (fig. 540). This structure in the early days of X-ray diagnosis was erroneously diagnosed as a fracture (Shepherd's fracture). I once had a case wherein this os trigonum led to a protracted action for damages.

Fractures of the *os calcis* are much more frequent. The symptoms are such as to differentiate this injury clearly from fracture of the malleolus or from a sprain. A special section will be devoted to this subject.

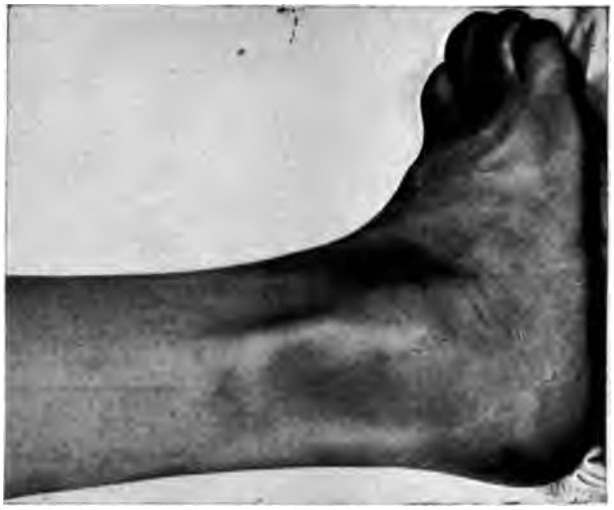


FIG. 544.—Dislocation of peroneal tendons, which project like cords over the external malleolus.

We should not be content with the diagnosis of **sprain** until a careful examination of the bones has yielded a negative result. Many a case of traumatic flat foot and years of persistent pain have been the result of an inaccurate diagnosis and an untimely permission to walk.

Laceration of the anterior tibio-fibular ligament may be mentioned as a special form of sprain. It is recognized by localized pain and probably by an effusion of blood above the ankle-joint, between the tibia and fibula.

Another injury is worthy of attention, despite its rarity. The patient himself probably hears a distinct crack at the moment of the accident and makes his own diagnosis of fracture. But neither palpation nor a skiagram reveal such a lesion. The tenderness is situated at the posterior edge of the external condyle—a position which is not the seat of pain in a sprained foot. If the peroneal tendons are, however, made tense, it will be seen that they move forwards, one after the other, over the malleolus, if indeed they are not already found there at the beginning of the examination.

The case is thus a typical **dislocation of the peroneal tendons** (fig. 544), and the crack corresponded to the moment in which the wall of the tendon-sheath compartment yielded to the sudden and sharp contraction of the muscle.

(2) INJURIES WITH DEFORMITY.

The diagnosis of sprain does not enter into consideration if, after an injury, the shape of the foot shows any deviation to one side or the other. The question then arises as to a **fracture** or a **dislocation** being the cause of the displacement, or as to the possibility of both being equally concerned in the injury.

We must first recognize the nature of the deformity. For this purpose both limbs are brought into the same position with the patella directed forwards. We then compare the direction of the axis of the leg and the instep on either side, as seen from the front, and the relations of the axis of the leg to the sole, as seen from the side. On inspection from the front we must note whether the long axis of the foot forms an abnormal angle with the axis of the leg, or whether it is displaced parallel to its normal position. On inspection from the side, we must test whether the axis of the leg strikes the foot too far forwards or too far backwards. If we are in doubt about any of these deviations, we should see whether we can cautiously rectify any indication thereof, or, on the other hand, whether we can increase it.

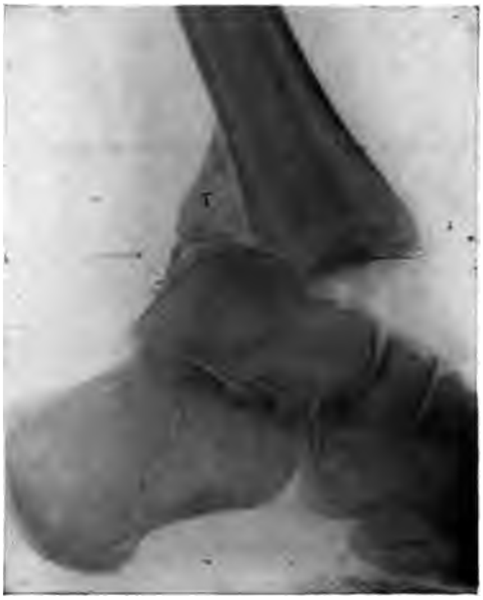


FIG. 545.—Bi-malleolar fracture with detachment from posterior edge of tibia (T), and backward displacement of foot (the tibia forwards).

The commonest displacement of the foot is *postero-external*, wherein the axis of the foot usually makes with the axis of the leg an obtuse angle open outwards. The foot thus has slipped backwards and outwards, and at the same time has become tilted somewhat outwards. The foot is usually in a condition of slight plantar flexion (fig. 546).

We start by palpating the lower end of the shaft of the tibia, because the displacement just described is often caused by a supra-malleolar fracture. We then proceed to the malleoli. The most frequent condition found consists of an abduction fracture thereof, i.e., a detached fragment from the internal malleolus, and a fracture of the fibula due to excessive bending. The astragalus is displaced outwards, and the upper fragment of the fibula rests directly upon it

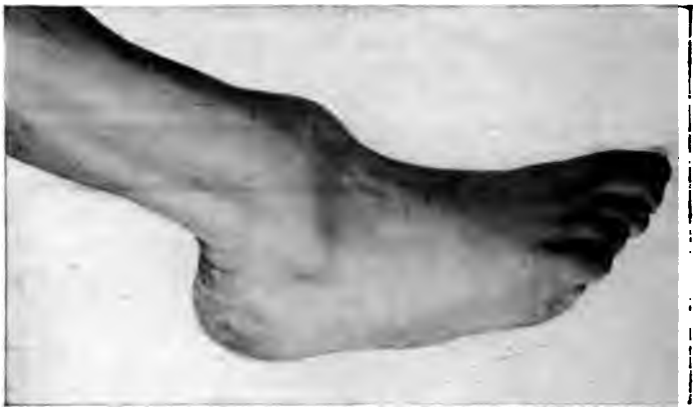


FIG. 546.—Dislocation of the foot backwards.

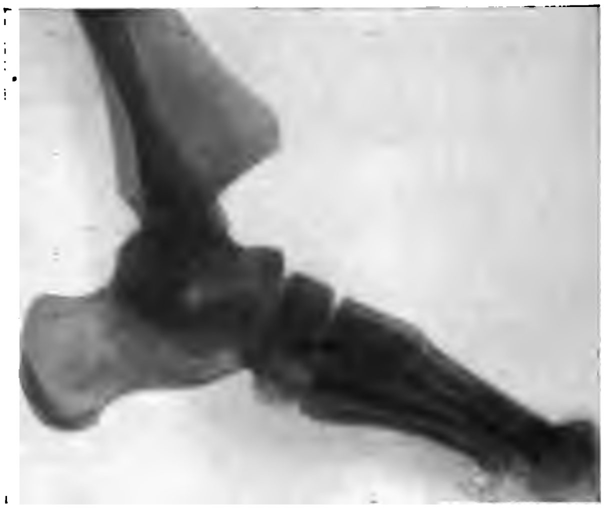


FIG. 547.—Incomplete posterior dislocation of the foot.

as shown in the skiagram. At the same time the foot is displaced backwards in relation to the leg, so that on a lateral view the tibia appears to project forwards, beyond the trochlear surface of the astragalus (fig. 547). Thus there is a **bi-malleolar fracture with subluxation of the foot backwards and outwards,** and displacement of the tibia forwards and inwards. The predominance of the one or other aspect of the deformity depends upon the nature of the injury. The more pronounced the displacement, and the less definite the fracture, the more justifiable is it to speak of a **dislocation,** without, however, being able to draw a hard-and-fast line between the two forms of injury.

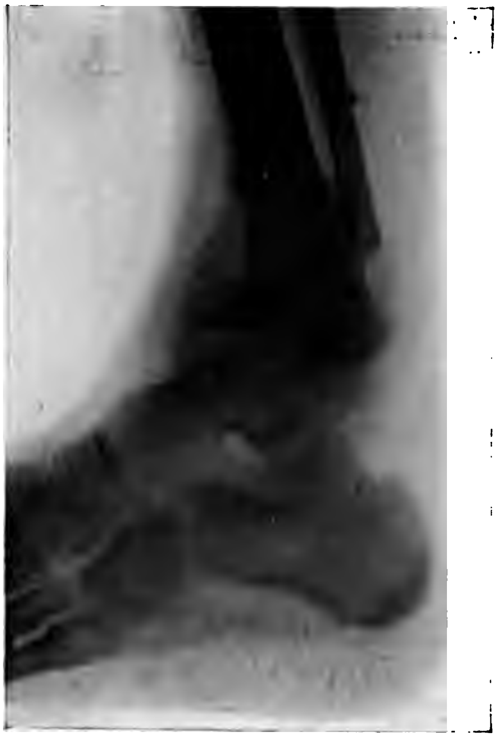


FIG. 548.—Bi-malleolar fracture, with displacement of the foot forwards (old case, so-called "Assyrian foot").

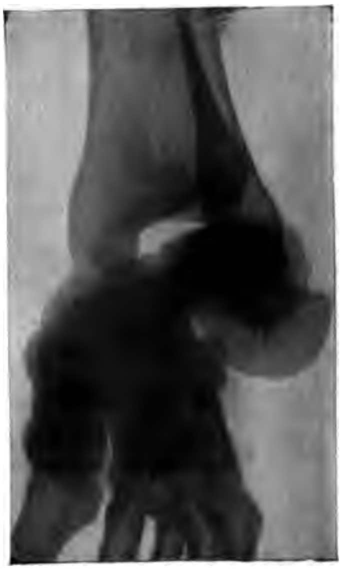


FIG. 549.—Dislocation of the astragalus. (Skiagram of fig. 550.)

If we feel both malleoli intact, through the skin, and also see the outline of the bifurcation of the malleoli projecting forwards (fig. 546), we at once diagnose a *pure dislocation.* We may, however, still be in error, because a fracture of the fibula may exist much higher up.

This occurred in the case illustrated in fig. 547. The patient, who was devoted to sleighing, knocked up against an obstacle with his heel, while the vis inertiæ of his tibia continued forwards.

We diagnose, in a similar manner, the very much rarer **dislocations** and **dislocation-fractures with displacement of the foot to the inner side and forwards.**

It is worth mentioning that the foot may be displaced *forwards* with the tibia resting on the posterior portion of the trochlear surface of the astragalus. This gives rise to a subsequent deformity which has been appropriately termed "*Assyrian foot,*" owing to its resemblance to Assyrian sculptures (fig. 548).

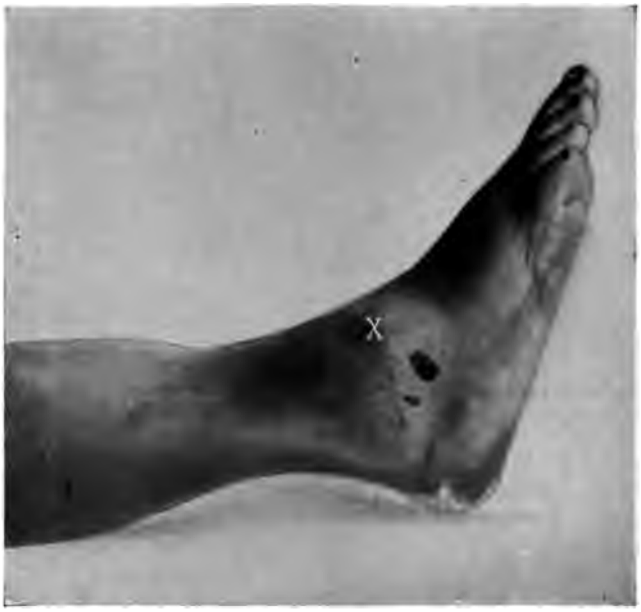


FIG. 550.—Dislocation of the astragalus forwards and outwards. X = projection of head of astragalus; necrosis of skin beginning (formation of vesicles).

We now come to a very different clinical picture of an injury, which may present itself in the same anatomical region. In this condition, which occurs after a severe injury to the foot, we find a round bony projection directly in front of the anterior edge of the tibia, or somewhat to its inner or outer side; the skin is tightly stretched over this projection, according to the position in which the foot is displaced in relation to the tibia, either forwards, inwards or outwards. These signs point to one diagnosis only, viz., **dislocation of the astragalus,** i.e., the disconnection of the astragalus from all its ligaments and its displacement under the skin. It does not matter, from the point of view of treatment, whether the bone is uninjured or actually broken—as sometimes happens. Fracture could only be detected clinically if crepitus is distinctly heard.

It is very important to make the diagnosis of dislocated astragalus as soon as possible, because the stretched skin over the displaced bone may become gangrenous within a few days, unless early treatment is applied.

If we find a striking displacement of the foot in relation to the leg, although nothing wrong can be detected in the malleoli, whose relations with the astragalus seem to be normal, the cause may still be a **dislocation below the astragalus.** Such dislocations are extremely rare, but they may occur in any direction. An intelligent inspection and palpation will easily decide whether the foot is displaced forwards, backwards or outwards.

CHAPTER CVI.

FRACTURE OF THE OS CALCIS.

A TYPICAL fracture of the os calcis cannot be mistaken for any thing else, and yet it is an injury which is frequently overlooked. It occurs either through the pull of the tendo Achillis—a laceration fracture—or through its compression between the astragalus and underlying surface—a compression fracture.

The term *laceration fracture* should be limited to the cases wherein the fracture involves the tuberosity of the os calcis, and then only if the line of fracture run parallel to the beam of the bone (fig. 551 *a*) ; or if its course is more oblique towards the plantar surface, the term should be limited to the cases wherein the fracture does not reach as far as the under surface of the bone. All the other fractures are *compression fractures*, whether they involve the body of the bone or its anterior process.

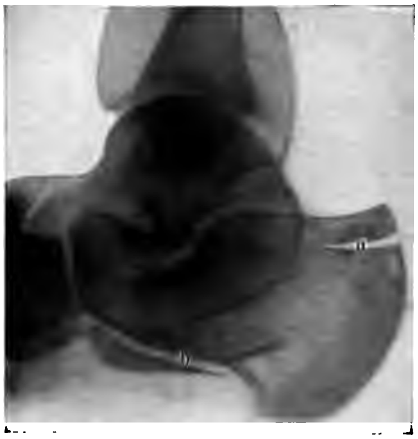


FIG. 551.—Double fracture of the os calcis. *a*, Laceration fracture ; *b*, compression fracture.

This does not, however, justify us in concluding that we can tell the form of the fracture from the nature of the injury. Both laceration fractures and compression fractures of the heel are usually due to falls on the sole from a height, and it occasionally happens that both forms of fracture occur in the same bone (fig. 551). Clinical examination and a skiagram render the differentiation of the two varieties very easy.

The clinical signs may be divided into five groups corresponding to five principal varieties of fractures of the os calcis.

(1) In fractures of the first variety, occurring after a fall from a height, we find that the foot is not lower than normal, that is to say, that the extremities of the malleoli maintain their normal distance from the ground. On the other hand, however, we are struck by a localized thickening at the lower end of the tendo Achillis, at the upper part of the tuberosity of the os calcis. On palpation the swollen area is found to be tender, but there is no tenderness on the under surface of the os calcis. We may possibly be able to obtain crepitus. Pressure in the axis of the leg is not painful. The patient is able to stand on his foot, and even to walk with a certain amount of pain. If ecchymosis occurs, it will be found on both sides of the tendo Achillis. In such a case the localized situation of the changes enables us to make the definite diagnosis of *laceration fracture*, which is very easily confirmed by a skiagram.

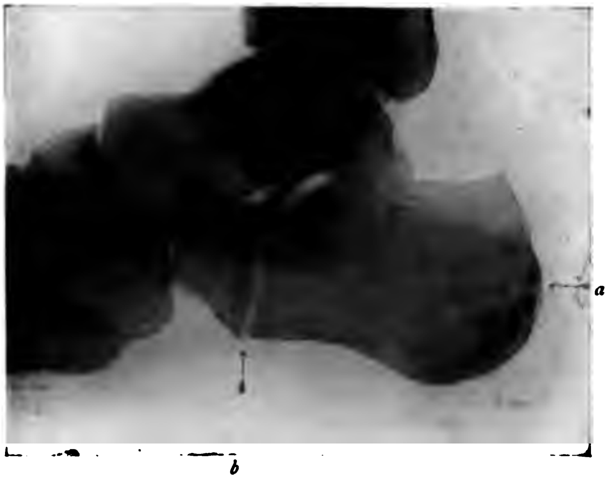


Fig. 552.—Double fracture of the os calcis. *a*, Fracture of the tuberosity of the os calcis; *b*, detachment of the anterior process.

(2) In other cases there is nothing abnormal to be felt at the tuberosity of the os calcis. The distance of the malleoli from the ground is normal, and the heel, as seen from behind, does not appear to be broadened. Nevertheless the patient avoids putting any weight on the injured foot. On palpation some slight thickening may be found, but there will be a special indication of pronounced tenderness

situated towards the tuberosity of the os calcis, or on the plantar surface, or towards the anterior process. Pressure on the sole of the foot in the axis of the leg is painful. The bruising usually occurs on the sole. In such a case we should think of a *compression fracture without marked displacement of the fragments.*

The skiagram shows either a certain amount of obliteration of the bony structure, from which we would conclude that a slight degree of crushing of the inside of the os calcis has occurred, or we may find distinct fissures running along the length of the bone (fig. 551 *b*) and transversely to it. Detachment of the anterior process is a special feature of this injury (fig. 552).

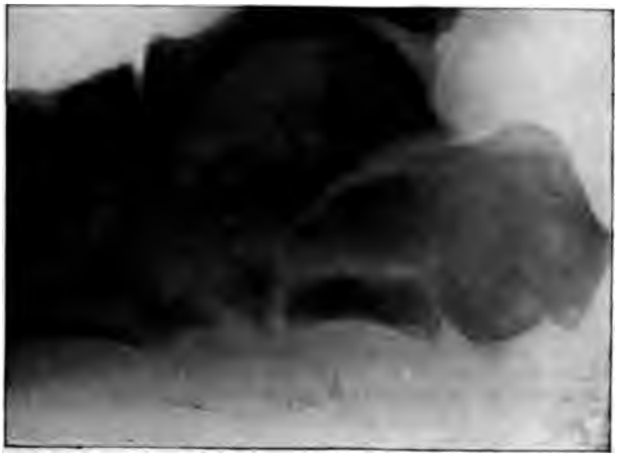


FIG. 553.—Severe crushing of the os calcis.

These cases are usually diagnosed at first as sprains or contusions. It is only when the patient attempts to put his foot to the ground in two or three weeks' time, and the pain still persists, that a more severe form of injury is thought of. Palpation will now reveal a distinct thickening of the os calcis by callus, even in those cases wherein nothing abnormal could be found on the first examination.

(3) The cases in the third group are much more easily recognized. The heel, as seen from behind, is evidently broadened from the very beginning, and the malleoli are lower than on the uninjured side. The os calcis is felt to be thickened, and is tender both on lateral pressure and on pressure in the axis of the leg. These are cases of *comminution fractures,* which are made up of fissures running lengthwise and also transversely. Obviously the more accurate details can only be made out from a skiagram. A tracing of the foot will show that it is widened about the heel. Patients are sometimes able to walk about even after this injury, although the pain is considerable; pronation and supination are particularly deranged.

(4) *Detachment of the sustentaculum tali*—a very rare condition—should be thought of if we find that the foot is definitely in a valgus position, and the region below the internal malleolus tender, without the signs of fracture of the malleolus or of the astragalus, or those of an ordinary compression fracture.

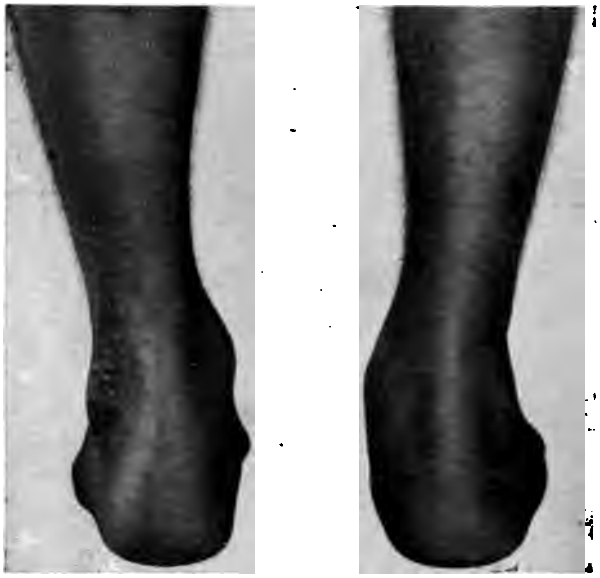


FIG. 554.—Old compression fracture of the right os calcis, treated as a sprain. The heel is broadened, the malleoli are low, and the position is one of slight valgus.

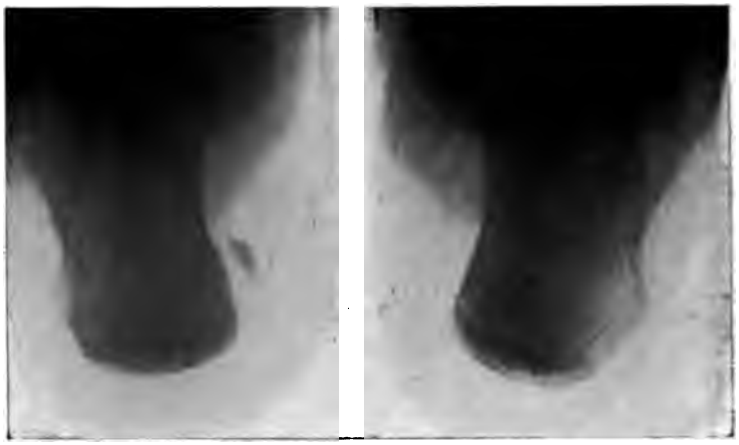


FIG. 555.—Detached fragment from inner side of os calcis.

(5) Finally, there may exist lateral detachments of bone, which are

only recognizable by the circumscribed pain on pressure and by X-ray examination from above, with the foot greatly dorsi-flexed (fig. 555).

The following scheme briefly summarizes the foregoing remarks on injuries about the ankle-joint :—

No striking deformity, at most a little swelling	Bone nowhere very tender, except at attachment of ligaments to one or other malleolus ; pain often persists, even when at rest. No pain on axial pressure.	.. ..	**Sprain of ankle.**
	Tenderness of bone transversely above joint (generally on tibia and fibula); pronounced pain on axial pressure	.. ..	**Supra-malleolar fracture without displacement.**
	Great tenderness of one or both malleoli at a more or less considerable distance above their extremities ; false mobility not always demonstrable, but most likely in the fibula ; no pain on axial pressure	.. ..	**Malleolar fracture without displacement.**
	Tibia and fibula not tender on pressure ; pain on axial pressure (by pressing os calcis on underlying surface) usually present ; pain on lateral compression of the os calcis	Malleoli at normal level ; pain over tuber calcanei	**Fracture of tuberosity of os calcis.**
		Ditto, but pain in body of os calcis	**Fracture of body of os calcis without displacement.**
		Malleoli abnormally low ; pain in body of os calcis	**Fracture of body of os calcis ; bones compressed.**
	Ditto, but pain on pressure in the astragalus and not in os calcis ; dorsi-flexion particularly painful	.. ..	**Fracture of astragalus.**
Foot displaced in relation to axis of leg.	Bone not tender ; bifurcation between malleoli can be felt under skin	.. ..	**Simple dislocation** (foot usually displaced backwards and outwards).
	Both bones tender transversely above the malleoli	.. ..	**Supra-malleolar fracture with displacement,** before the age of 14 usually a **separation of epiphysis** (foot generally displaced backwards and outwards as in dislocation).
	Bone tender above tip of malleolus (tibia or fibula) or above the malleolus (fibula); sometimes accompanied by an actual dislocation	.. ..	**Fracture of malleolus with displacement** (foot generally displaced as above, rarely forwards).
	Malleoli not tender ; bifurcation between malleoli abnormally easily felt; a round body projects, usually forwards, under the stretched skin	.. ..	**Dislocation of astragalus** (often combined with fracture).
	Malleoli normal on palpation ; the bifurcation between them not very easily felt ; considerable displacement of foot below astragalus ; head of latter to be felt	.. ..	**Dislocation below astragalus** (occurring in various directions).

CHAPTER CVII.

INJURIES TO THE FOOT, IN FRONT OF THE ANKLE-JOINT.

WE need not occupy any time over such rare injuries as *dislocations of Chopart's or Lisfranc's joints.* The displacement is so striking that the diagnosis can be made from anatomical considerations, unless the swelling is very great, in which circumstance a skiagram will be necessary.

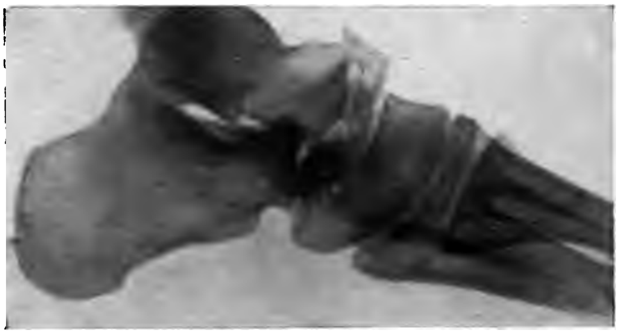


FIG. 556.—Compression fracture of scaphoid.

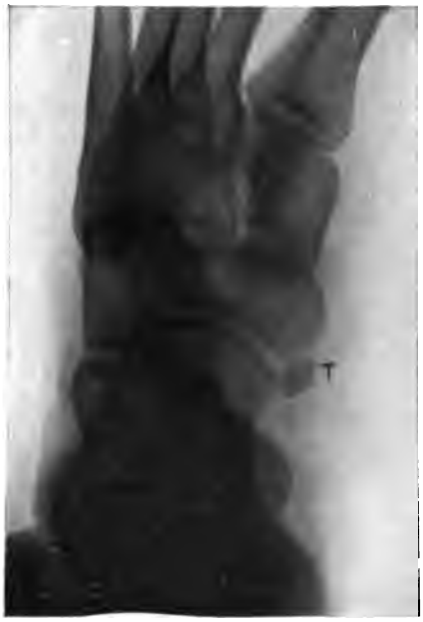


FIG. 557.—Os tibiale externum (T).

More interest attaches to certain trivial, but not infrequent, injuries which are quite characteristic, and which were previously overlooked or incorrectly diagnosed. Owing to recently acquired knowledge, these injuries may now be recognized by their clinical signs.

(1) **Compression Fracture of Scaphoid.**—If a person falls from a height on to his toes, the force is to a great extent gathered up by the scaphoid and transmitted to the astragalus. If the scaphoid is not sufficiently resistant, it becomes compressed, and, so to speak, is squeezed out of the skeleton of

the foot. It then gains the dorsal surface, and can be distinctly felt through the soft parts (fig. 556).

In the female sex a Wormian bone, described by Gruber as the os tibiale externum, is often found on the tubercle of the scaphoid (fig. 557). Ignorance of this abnormality has led to the erroneous diagnosis of fracture, when the ligaments have been torn. The condition, when looked upon as a fracture, has been termed "dance injury." The presence of the Wormian bone on both sides ought to be a safeguard against errors.

(2) **Fracture of a metatarsal bone** is much more frequent. If a heavily laden soldier, wearied by many hours' marching, begins to relax, and fails to impart the necessary elasticity to his steps by the proper use of the muscles of his foot, it is very easy for the metatarsus to become overweighted, and for one of the bones—usually the second—to crack. The symptoms thus caused: spontaneous pain, tenderness on pressure, and swelling, were previously attributed to inflammatory changes in the soft tissues, until it was shown by X-rays that they were really caused by a subperiosteal **metatarsal fracture,** usually without any dislocation.

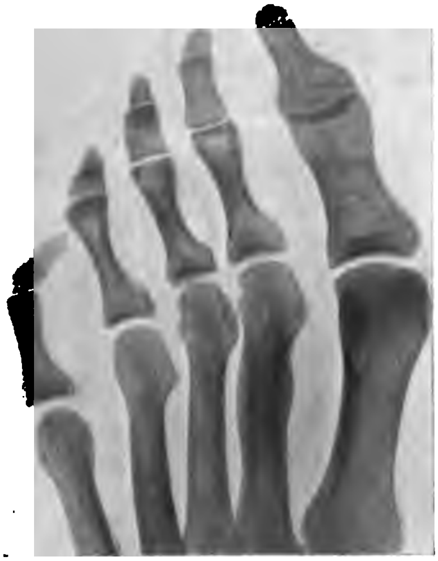


FIG. 558.—"Swollen foot." Old callus.

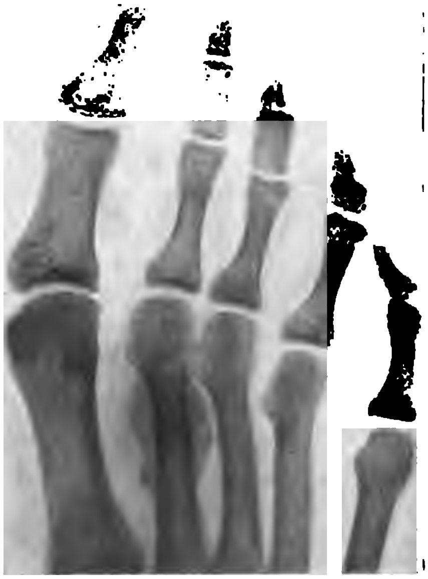


FIG. 559.—"Swollen foot." Recent callus.

Sometimes the skiagram which is taken forthwith reveals no change at all, because the fissure in the bone is purely subperiosteal. But in a few weeks' time slight callus is visible in the skiagram.

The following is an illustrative case :—

A slimly-built recruit showed the well-known signs of "swollen foot" in the neighbourhood of the second right metatarsal after a long march. The skiagram, which was taken immediately, revealed nothing ; but a diagnosis of fissure was nevertheless made. A subsequent examination in a few weeks revealed definite callus. In a few months' time the young man had to resume his military duties, and the symptoms returned in his left foot after a long march with full equipment. On this occasion also the skiagram was negative, but examination a few weeks later showed the condition illustrated

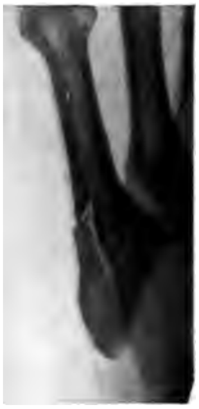


FIG. 560.—Fracture of tuberosity of fifth metatarsal.

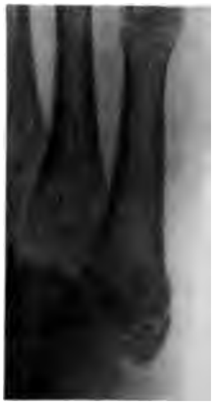


FIG. 561.—Epiphyseal line at base of fifth metatarsal (from lad aged 14).

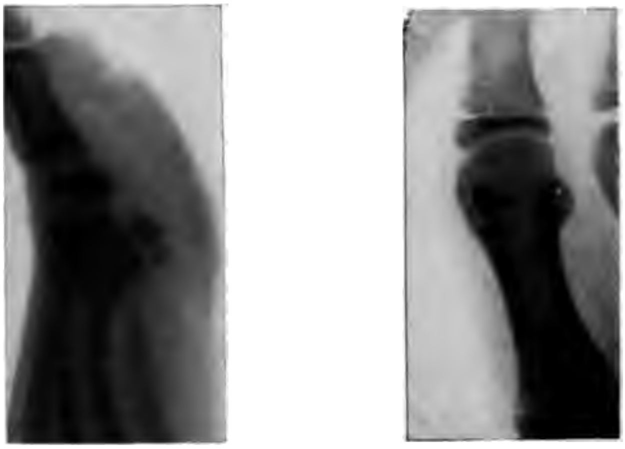

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FIG. 562.—Fracture of sesamoid bone, seen from below and from the side.

in figs. 558 and 559. The second metatarsal bone of the right foot, which was injured first, presented a spindle-shaped thickening which represented the remains of callus. The second metatarsal of the left foot presented well-developed recent callus. Before entering military service the patient had done much mountaineering without any resulting injury to the bones of his foot, thus showing that the military conditions are really responsible, i.e., the heavy equipment and the forced marching of the weary soldier when his muscles are exhausted.

(3) **Fracture of the tuberosity of the fifth metatarsal bone** is also a characteristic injury of the metatarsus. It can be diagnosed clinically, but is easily overlooked. It may be caused by direct violence, and probably also by the contraction of the peroneus brevis; it has the appearance of a detached fracture (fig. 560). This fracture must not be confused with the separation of the epiphysis (fig. 561), which occurs in this situation between the ages of 12 and 14, as described by Kirchner and Iselin.

(4) The possibility of fracture of a sesamoid bone (fig. 562) will be suggested by localized pain on pressure over the sole, and by painfulness of the movements of the toes.

We need not enter into details of the numerous and varied fractures and dislocations which may occur about the heads of the metatarsal bones and the toes. Pressure and traction on the separate toes will always show which of them is involved; but a skiagram will be required for further details.

CHAPTER CVIII.

INFLAMMATORY DISEASES OF THE FOOT.

A.—TARSUS.

INFLAMMATORY diseases of foot may be termed *podarthritis* on the principle that inflammatory processes of the wrist are sometimes called *cheir-arthritis*. A general term of this nature may be justifiable in the case of the wrist, because of the small amount of bony structure which is involved, but in the case of the foot the diagnosis must be more definite.

We differentiate :—

(1) ACUTE DISEASES.

It is usually easy to determine whether an acute inflammation involves the ankle-joint, or is situated more forwards in Chopart's or Lisfranc's joint. In the vast majority of cases the ankle-joint is alone involved, or at least is the principal seat of the affection.

The nature of these acute inflammations is similar to those which occur in the shoulder and knee, and as also happens in these joints, the original site of the disease is frequently in the adjoining bone, and not in the joint itself.

(2) CHRONIC INFLAMMATION.

Tubercle is, as always, the most important of the chronic inflammations. Adults are more frequently affected with tuberculosis of the ankle than any other joint. The patients usually complain of pain, of a few weeks' or months' duration, about the ankle. There is consequently some lameness. There is frequently no evident change at this stage; the only sign of disease may be a certain amount of tenderness in the region of the capsule of the upper part of the ankle-joint. A skiagram may reveal nothing but a striking transparency of the bone (fig. 563)—osteoporosis—due to the disappearance of lime salts. If the disease is somewhat more advanced, there may be swelling of those portions of the capsule accessible to palpation, or there may possibly be a para-articular abscess. The grooves on either side of the tendo Achillis are very frequently obliterated at this stage, and the tendon itself may appear to lie in a depression (fig. 565). The skiagram will show that the articular surfaces are partially eaten away. The mutual approximation of the bones indicates that the cartilage has already to some extent disappeared. It may be possible to recognize individual areas of disease in the bone, for the diagnosis of which the reader is referred to the chapters on the hip and knee-joints.

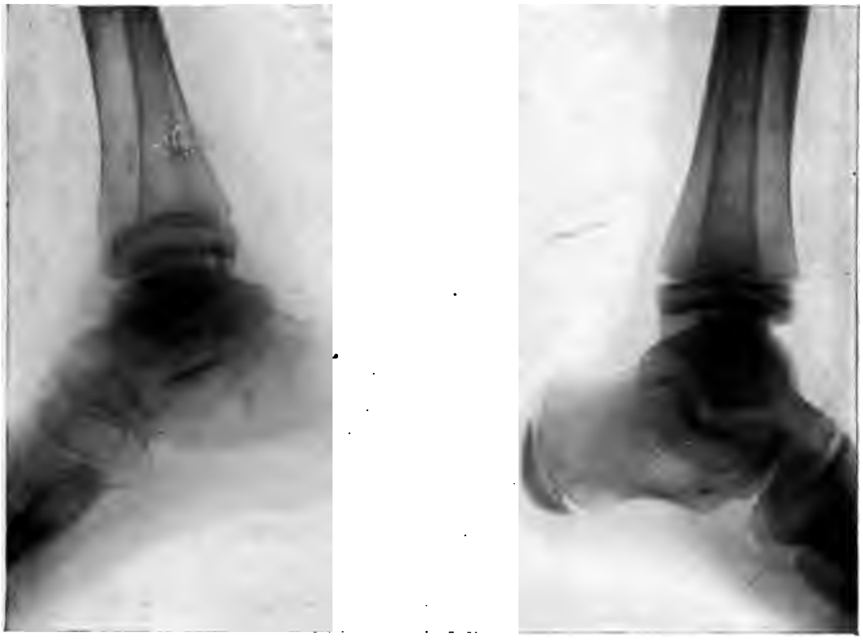
When the capsule is swollen the condition may be mistaken for **gummatous disease** or chronic **gonorrhœal** effusion. The former is less painful and the latter more painful than tubercle usually is.

If the disease process has broken through externally, it favours the diagnosis of tubercle, as against rheumatism or gonorrhœal arthritis. It is not, however, any contra-indication of syphilis; on the contrary, it suggests tertiary disease if the appearance on the skin is not that of a fistulous orifice, but that of a sharply marginated ulcer.

In the tropics we may be confronted with "madura foot," a disease allied to *actinomycosis* which may give rise to a clinical picture similar to tubercle or syphilis.

In the cases hitherto discussed the principal feature has been involvement of the ankle or its neighbouring joints. But we frequently find that lameness and pain in the foot may arise, although the *movements of the joints are perfectly free.* Accurate examination will show that the tenderness is not in the capsule of the ankle-joint, or of Chopart's joint, but that it is in the lower end of the tibia or the os calcis, or in rarer cases in the scaphoid or cuboid.

Tubercle of the lower end of the tibia may occur in one of three forms: (1) A simple central abscess of the bone, with diffuse thickening of the cortex; (2) a lesion with a spongy sequestrum situated quite close to the joint; (3) small areas of granulation. The last applies to **tubercle of the os calcis,** which is rather frequent, and which is usually situated in its posterior half. We generally find one or more abscesses surrounded by sclerosed bone, which often contains large spongy sequestra. The disease sometimes remains within the os calcis for years, and only becomes manifest from time to time by fresh inflammatory exacerbations. This intermittent course and the skiagram render it easy to differentiate tubercle from **sarcoma,** which has occasionally been observed in the os calcis.



Left (diseased) side. Right side.

FIG. 563.—Early stage of tubercular synovitis of the left ankle-joint. The skiagram only shows great osteoporosis.

It is very important not to confuse early tubercle with the so-called **Achillodynia.** This term indicates a painful inflammation of the mucous bursa between the tendo Achillis and the os calcis—due either to rheumatism, gout, or gonorrhœa. It may also come on, after fatiguing marches, and is thus particularly liable to occur in soldiers, or in mountaineers, who have no kind of predisposition thereto.

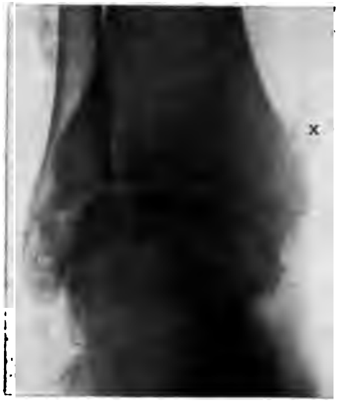


FIG. 564. — Tubercle of ankle. Narrowing of cartilage. Bone eaten away. Lesion in external malleolus. New bone formation at X.

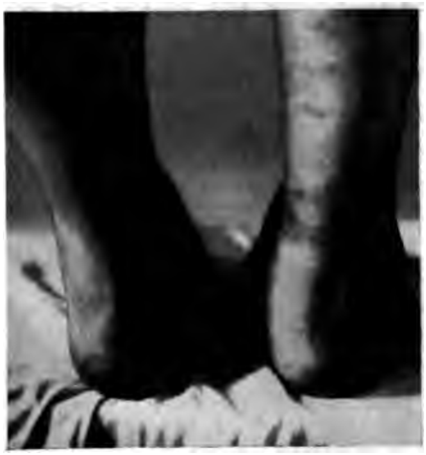


FIG. 565.—Tubercle of right ankle. Tendo Achilis looks like a furrow between the two lateral swellings of capsule.

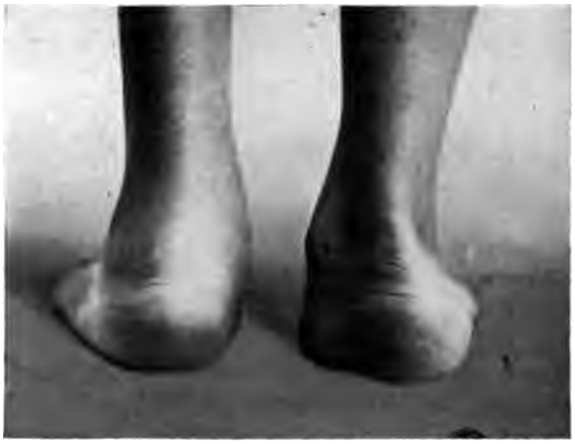


FIG. 566.—Tubercle of left ankle (both feet are flat). The grooves on either side of tendo Achilis are obliterated on the left.

Affections of the *bursa subcalcanea*, and of the *bursa Achillea posterior*, which lies on the tendo Achillis may occur under similar circumstances. Traumatic or painful inflammatory swellings are sometimes found in the *tendo Achillis itself*, after long marches.

The pain produced by the sub-calcaneal bursa has been incorrectly termed "talalgia." "Calcanalgia" is a more correct designation, but "pternalgia" is quite superfluous.

B.—METATARSUS AND TOES.

If the metatarso-phalangeal joint of the great toe becomes tender on pressure, inflamed and spontaneously painful, a typical **attack of gout** is obvious.

It is of some diagnostic interest to recognize that a typical attack may occasionally be followed by signs of visceral gout. In rare cases the attack starts with visceral manifestations, to be followed by a typical seizure in the great toe. I have, for instance, seen a gouty subject, who had been free from attacks for ten years, suffer successively from angina, trigeminal neuralgia, gouty seizure in the foot, non-purulent urethritis, proctitis, sciatica, pneumonia and nephritis.

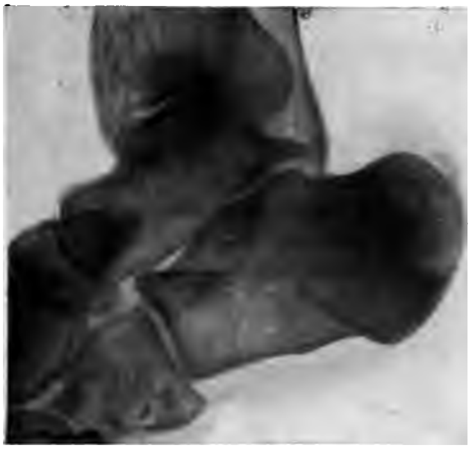


FIG. 567.—Tubercle of os calcis (superficial lesion in bone, with sequestrum).

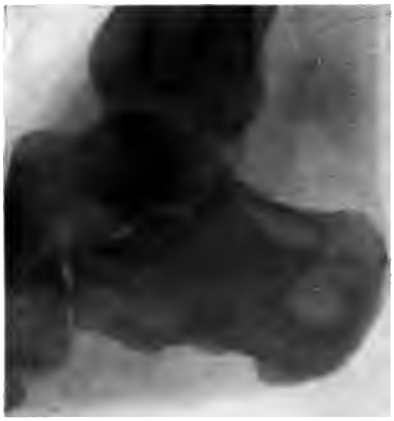


FIG. 568.—Tubercle of os calcis (deep abscess in bone).

Acute phlegmonous processes in the neighbourhood of a **hallux valgus** are generally due to suppuration in the bursa over the head of the metatarsal bone. These abscesses in the foot, which are situated under callosities, often assume the shape of a stud, just as they do in the hand. They really consist of two abscess cavities, the one being under the *epidermis*, the other more deeply situated under the *skin*, the two intercommunicating by a narrow opening.

If a toe exhibits signs of intermittent bluish-red congestion, with pains not limited to the discoloured area, but which may even extend to the leg, we should think of **commencing gangrene** in elderly or diabetic patients.

The considerations which apply to **dactylitis** of the hand and fingers are also applicable to **chronic inflammatory processes of the metatarsus and toes.** The first metatarsal bone suffers most frequently.

CHAPTER CIX.

DEFORMITIES OF THE FOOT.

Most deformities of the foot are so easily recognized that difficulties in diagnosis hardly ever arise. We shall therefore only refer to a few points which occasionally perplex beginners.

(1) FLAT FOOT.

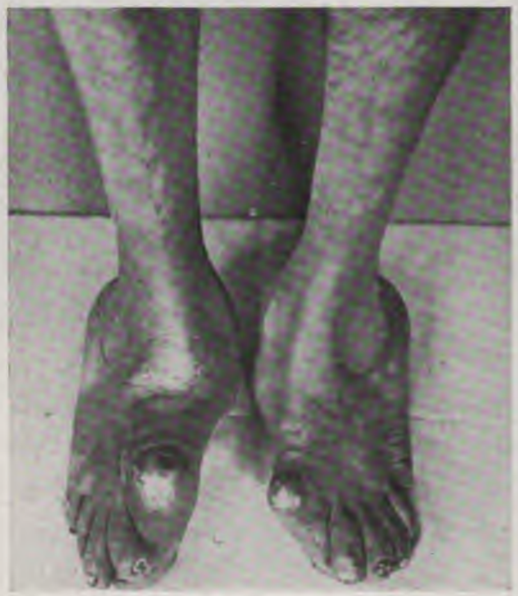


Fig. 569.—Flat foot (valgo-planus) of severe type with hallux valgus.

The frequency with which a valgus foot and a flat foot are combined has given rise to the impression that both deformities are of the same significance — an error which has led to much bad treatment.

Pes valgus, or everted foot, is characterized by the inclination of the os calcis outwards, i.e., it is not directly in the line of the axis of the leg, but forms an obtuse angle with it, as seen from the outer side. This angle disappears as soon as the foot is placed upon a correspondingly inclined plane (fig. 570). The foot becomes *flat* — *pes planus*—when the arch sinks, and the anterior part of the foot at Chopart's joint becomes abducted, so that its axis deviates externally from the perpendicular to the line connecting the malleoli (fig. 571).

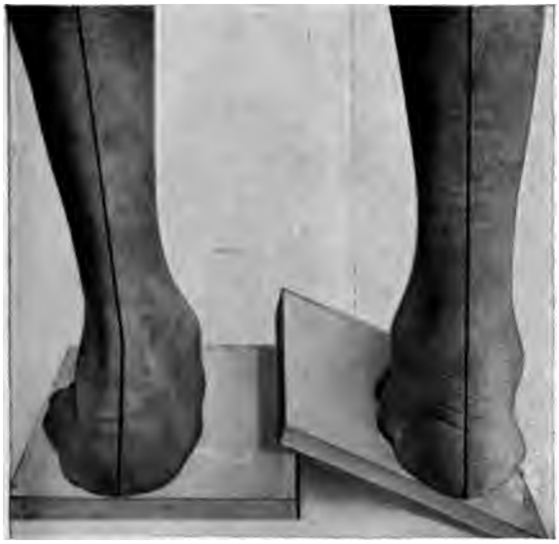


FIG. 570.—Bilateral pes valgo-planus. On the right side the valgus position has disappeared, because foot is resting on an inclined plane.

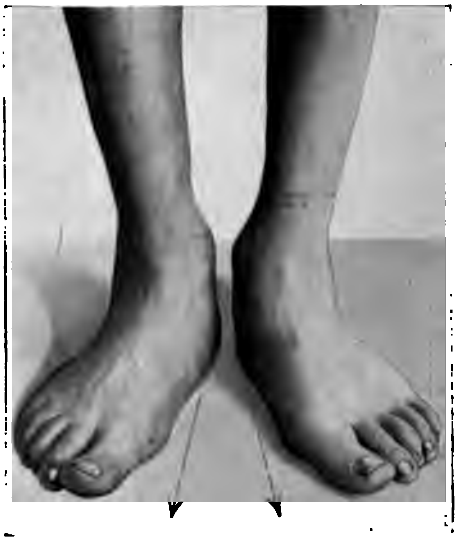


FIG. 571.—Same case from the front. External deviation of the anterior part of foot; the arrows indicate the normal positions of the inner margins of foot.

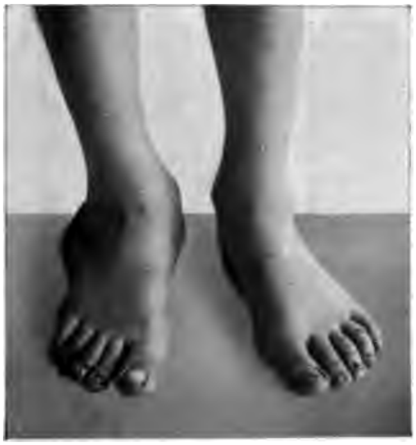


FIG. 572.—Pes valgo-planus on right side after traumatic division of the tibialis posticus tendon.

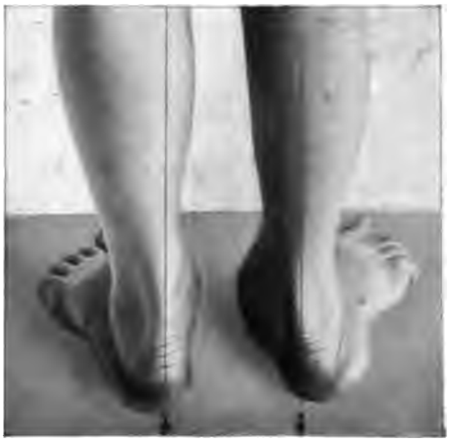


FIG. 573.—Same case from behind,

A flat foot in the valgus position, with these signs, is obvious even to a lay observer, especially if the shape is as grotesque as in fig. 569. In such cases, the impressions made by the sole of the foot will be of the character illustrated in figs. 576 and 577. These severe cases do

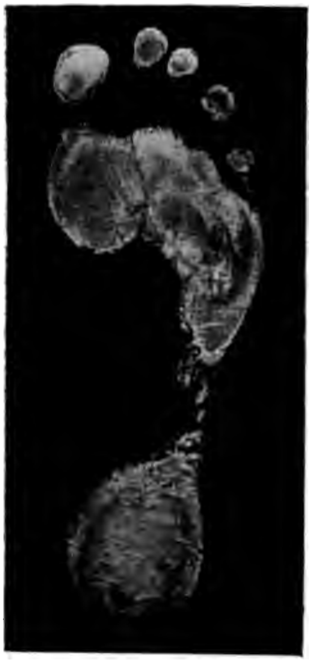


FIG. 574.—Normal foot.

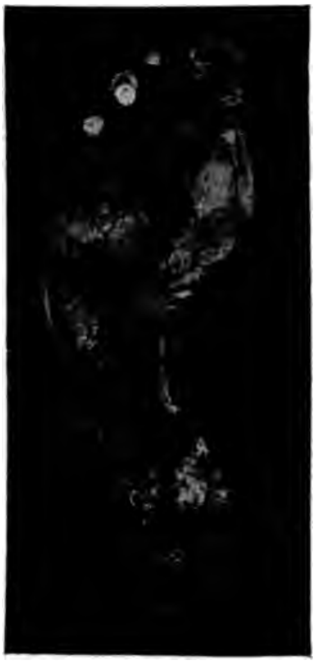


FIG. 575. — Commencing flat foot.

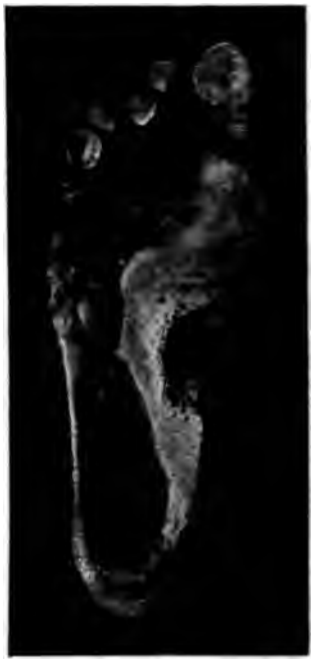


FIG. 576.—Moderate flat foot

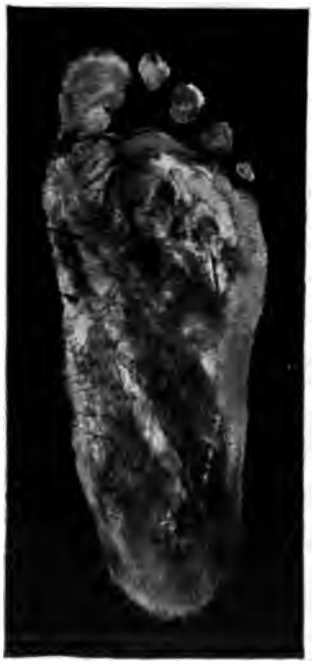


FIG. 577.—Severe flat foot.

FIG. 578.—Pes calcaneus.

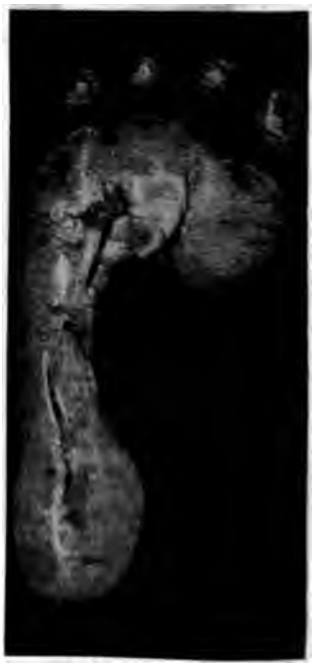


FIG. 579.—Pes excavatus.

not, however, possess as much diagnostic interest as those wherein the patient complains of pain in various places on his foot, without the presence of any definite flattening of the arch. But careful inspection will often show that the heel is turned somewhat outwards and that the anterior part of the foot has undergone some lateral deviation. The impression of the sole may nevertheless be almost normal, or at most show a somewhat wide connection between the heel and the balls of the toes (figs. 575).

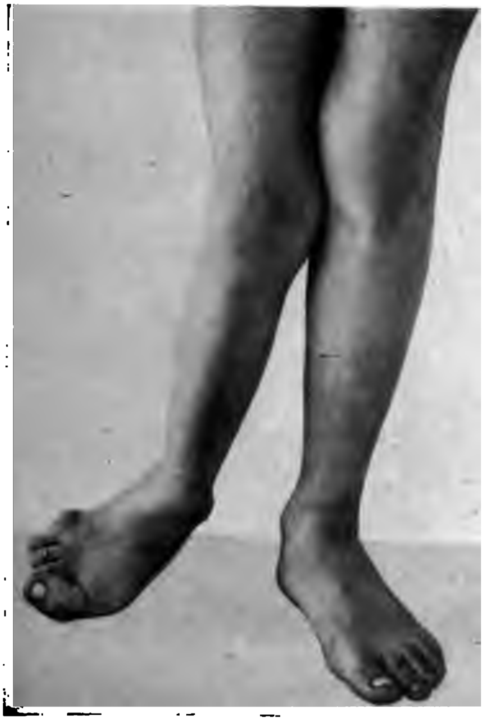


FIG. 580.—Congenital absence of fibula, with pes valgus, in girl aged 8 years.

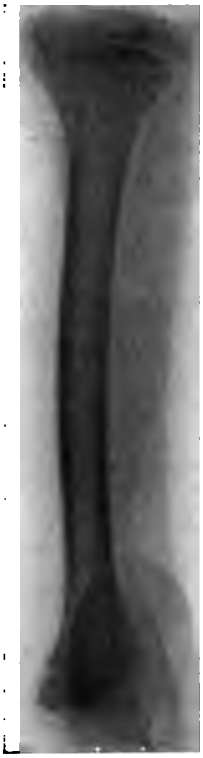


FIG. 581.—Congenital absence of fibula; skiagram of fig. 580.

The pain is sometimes localized to definite spots, i.e., astragalo-scaphoid joint, the head of the astragalus, the internal side of the scaphoid and the area in front of and below the external malleolus. In other cases the pain is more diffuse, extending over the whole tarsus, or radiating forwards between the metatarsal bones. The pain is very sharp on standing, it is less severe on walking, and it disappears rapidly on resting. Well-fitting boots relieve it; low soft shoes increase it. Occasionally the pains are felt as much in the calf as in the foot, or even more so. They may even be felt in the thigh.

If one neglects to make an examination in this stage, or does so only cursorily, such an unsatisfactory diagnosis as talalgia or metatarsalgia, &c., is apt to be given.

If the pains become so severe that the patient contracts all the muscles in order to fix the joints of the foot, the term *spastic flat foot* is employed—or incorrectly, *inflammatory* flat foot.

It may resemble a commencing tuberculosis in this stage. Flat foot may accidentally coincide with tubercle. If the first examination is not decisive, the patient must be instructed to rest for two or three weeks. The pain of flat foot will then disappear, but that of tubercle will either persist or only abate in a slight degree.

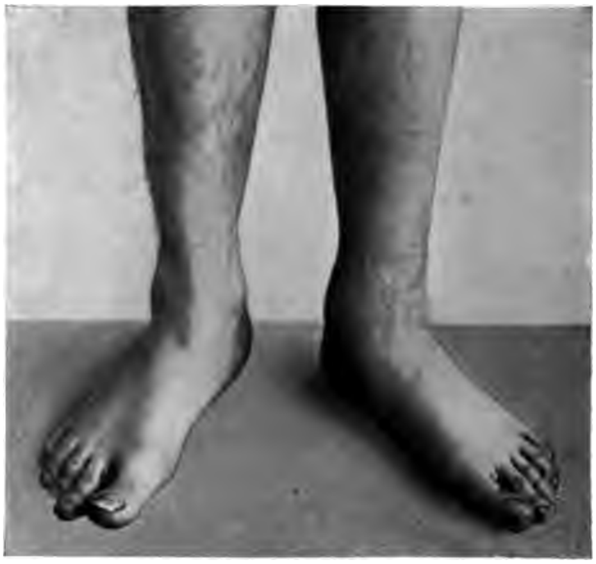


FIG. 582.—Habit contracture of left foot in valgus position, after a healed fracture of fibula without dislocation.

Errors of diagnosis, such as rheumatism, neuralgia, &c., are not likely to be committed even by the inexperienced, if the patient is a young person whose occupation demands constant standing. On the other hand flat foot is frequently overlooked in corpulent women at the climacteric period.

The increased body weight which often sets in at this age puts too great a strain on the slender bony structure of a woman's foot. It therefore sinks downwards and inclines outwards. The pains of flat foot supervene, and they are attributed to rheumatism, neuritis, to varicose veins, which are usually evident, and if they are not, to "deep varicose veins" which are usually discovered *ad hoc*. The fact that the pain is felt in the calf muscles is the basis of this last assumption.

A valgus foot or a flat foot, resulting from an *injury* (fracture of the malleolus, os calcis, or a metatarsal bone) often remains misunderstood for a considerable time, because the foot does not assume the classical picture of flat foot from the beginning. On the other hand, pes valgus may arise as "habit contracture" after an injury without any deformity of bone (fig. 582).

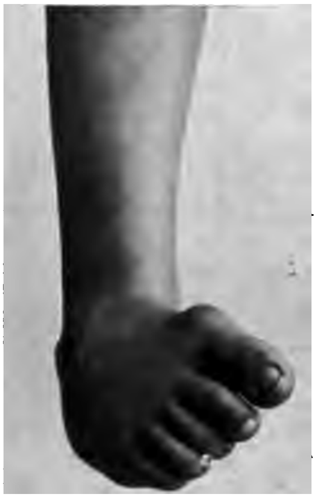


Fig. 583.—Congenital club foot (simple form).

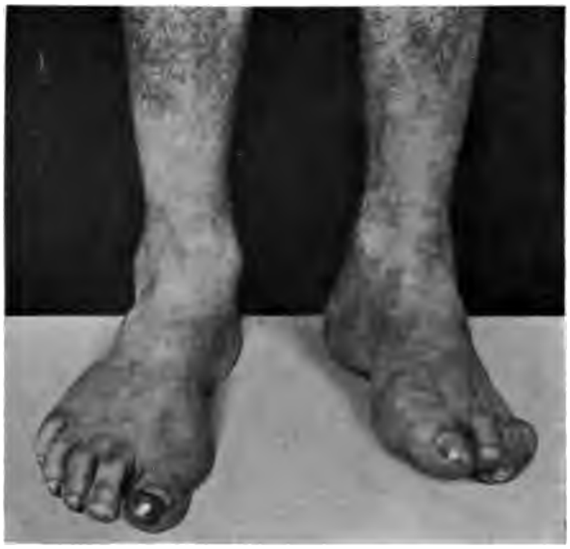


Fig. 584.—Habit contracture of left foot, assuming a club foot posture, after a sprain and completely healed fracture of posterior process of astragalus.

Paralytic flat foot as a symptom of paralysis, especially of infantile paralysis, is easily recognized. It resembles the flat foot which results from division of tendons (tibialis posticus, figs. 572 and 573).

Congenital absence of the fibula is suggested by a striking valgus posture with shortening of the limb and curving of the tibia forwards and inwards. Palpation will show that the external malleolus is absent. The tibia usually presents a scar-like stripe, running lengthwise (figs. 580 and 581). This mal-development is frequently bilateral and the fibula is absent, either completely or partially. The toes are sometimes quite perfect, at others they are imperfectly developed towards the little toe. The so-called *Volkmann's subluxation of the foot outwards* constitutes a slight degree of this deformity.

(2) TALIPES, PES EQUINUS, PES CAVUS, PES CALCANEUS.

These deformities are so distinctive where their characters are once known, that we may be content with a few typical illustrations. The *cause* of these deformities is, however, of importance, from the therapeutic standpoint. Whereas in flat foot the mechanical and rachitic changes predominate over those of paralytic or congenital origin, the deformities at the head of this section are mainly due to congenital or paralytic causes, rarely to injuries, and never to rickets.

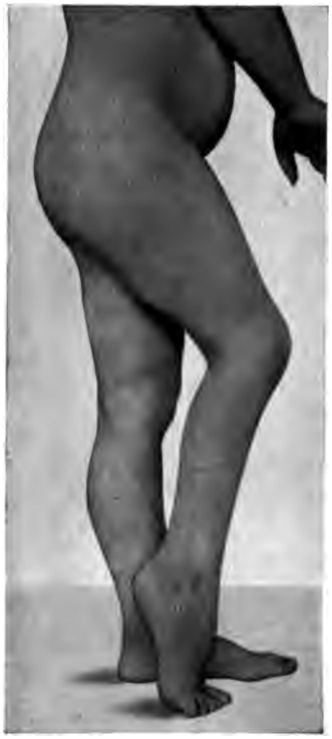


FIG. 585.—Paralytic pes equinus. (After infantile paralysis).

The first question in regard to **equinus** must be in regard to its *congenital* or *acquired* origin. The history usually supplies the answer. If not, we may assume a paralytic or acquired origin if there is coldness or blueness of the foot, and obviously also if definite paralysis exists. Atrophy of the calf muscles is of itself not conclusive evidence, because this may also arise in course of time from want of activity of certain muscle groups as a result of congenital club foot, and may indeed reach a very considerable degree. Neither does the unilateral or bilateral existence of the deformity give any information on this point, because the congenital and the acquired forms may both affect either one or two sides. But if it once be ascertained that the talipes is of the paralytic variety, certain important conclusions as to the *cause of the paralysis* follow therefrom.

Unilateral talipes with flaccid paralysis is usually due to acute anterior poliomyelitis, but may exceptionally be the result of spina bifida.

The latter cause is suggested by the simultaneous existence of disturbances of sensation and incontinence of urine. If no swelling is evident on the back, a spina bifida occulta should be looked for. Similarly, bilateral talipes with flaccid paralysis should suggest some congenital defect in the lumbar cord.

Unilateral talipes combined with spastic paralysis is due to infantile cerebral paralysis, but is in exceptional cases the result of an injury to the brain (see false meningocele). If both feet are affected with talipes and spastic paralysis, we may assume that the cause is a form of spastic diplegia known as Little's disease.

The spastic forms of club foot are classified as acquired, although their ultimate cause is congenital. The development of the deformity does not, however, occur until post-fœtal life.

One form of talipes deserves special mention because it plays an important part in the consideration of accidents, i.e., the so-called *habit contracture.* If for some reason or other, generally an injury, the movements of the ankle and Chopart's joint become painful, these joints are held rigid by muscular fixation so that the foot does not yield at all on walking, but is planted down stiffly on its outer edge, as in talipes. If the patient is very sensitive to pain, or has neuropathic tendencies, or if there be a question of compensation involved, this posture may persist after the disappearance of the pain or after the recovery from the injury; it becomes a *habit contracture.*

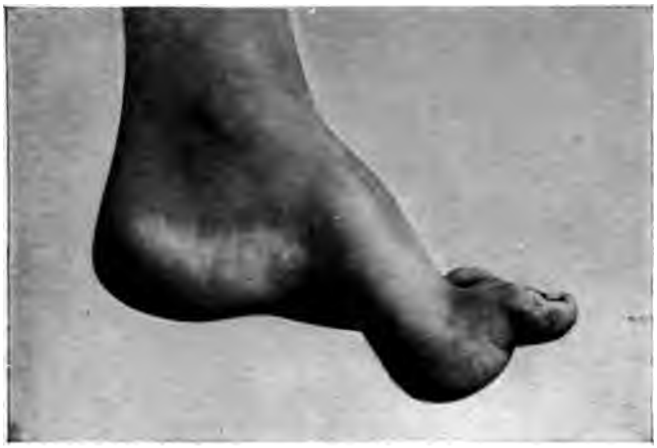


FIG. 586.—Paralytic pes cavus in a case of spina bifida.

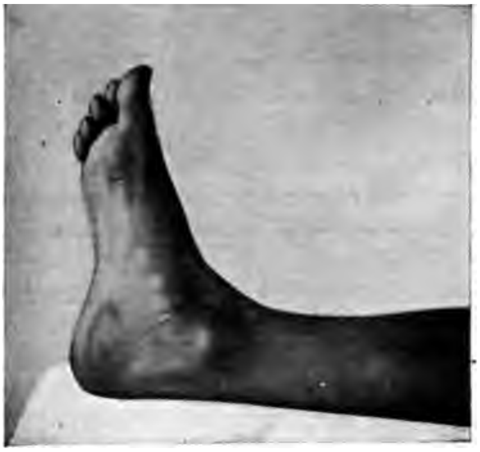


FIG. 587.—Pes calcaneus of slight degree, congenital origin.

I have seen a habit contracture of this kind come on after a contusion of the foot. Although there was not the slightest anatomical change, the foot was always held in the talipes position on walking.

The muscles of the whole limb were somewhat atrophic, and there was some cyanosis due to the deficient muscular activity. The condition had been present for three years owing to the protracted legal proceedings.

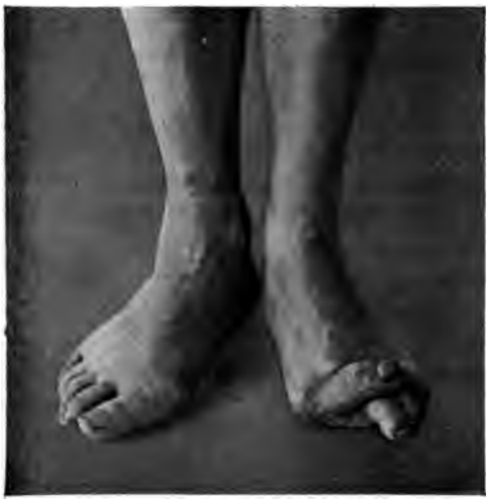


FIG. 588.—Hallux valgus of various degrees.

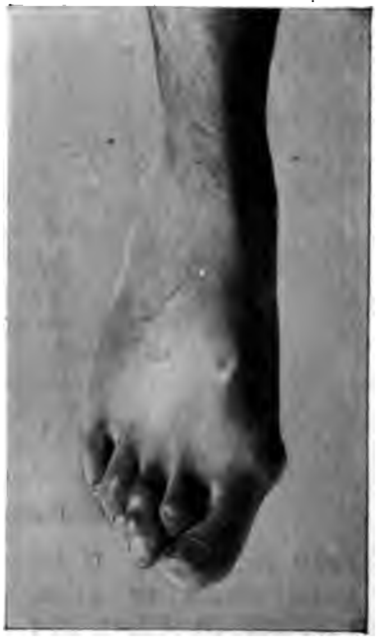


FIG. 589.—Hammer-toe (second).

The preceding remarks on ordinary talipes apply also to **pes equinus, pes calcaneus** and **pes cavus.** These varieties may be either congenital or acquired, and in the latter case are usually of paralytic origin. Bilateral pes cavus and sometimes also pes calcaneus may gradually develop in advanced infancy or at puberty, without any definite ascertainable cause.

They are probably due to some congenital disturbance of the co-ordinating power of the various muscle groups, and may be very slight signs of some hereditary maldevelopment of the spinal cord, such as defective development of certain anterior horn cells.

Some cases of congenital club foot, may probably be due to the same cause—apart from definite deformities. Most cases of hammer toe probably arise in the same manner.

The worst cases of pes calcaneus come on many years after tenotomy of the Achilles tendon in infancy—a proof of the effect of muscular action on the shape of the skeleton of the foot (fig. 593).

Fig. 585 shows that pes equinus may be mistaken at first sight for hip disease.

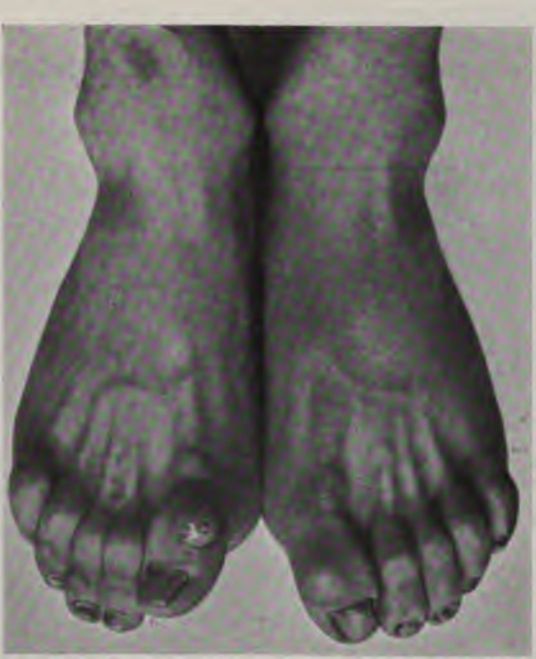


FIG. 590.—Universal hammer-toes (in a position midway between extension and flexion because the disturbance of muscular equilibrium is not associated with any special paralysis).

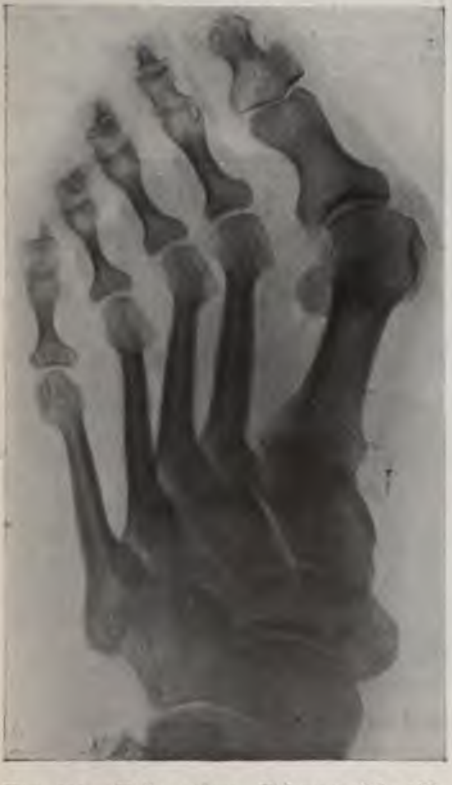


FIG. 591.—Hallux valgus. Skiagram of fig. 588.

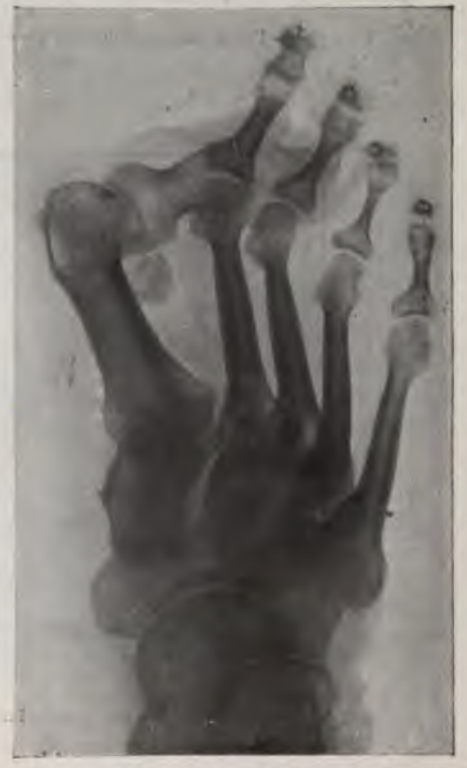


FIG. 592.—Hallux valgus. Skiagram of fig. 588.

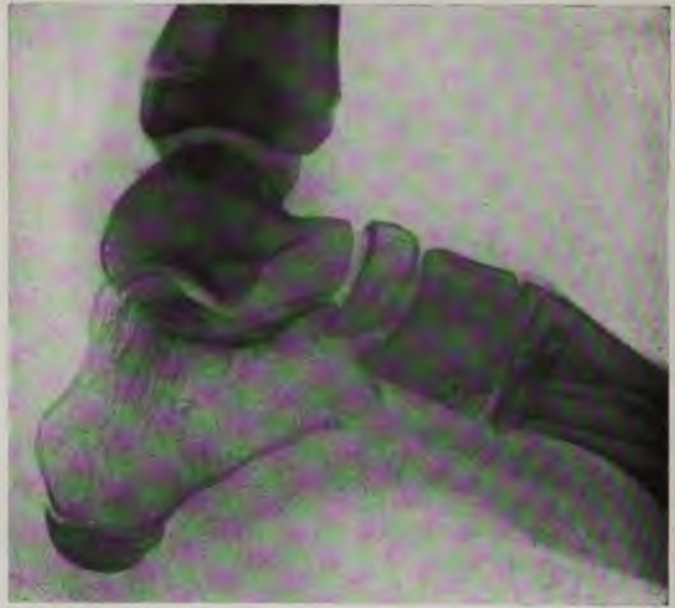


FIG. 593.—Pes calcaneus, fourteen years after tenotomy of the tendon of Achilles for spastic pes equinus.

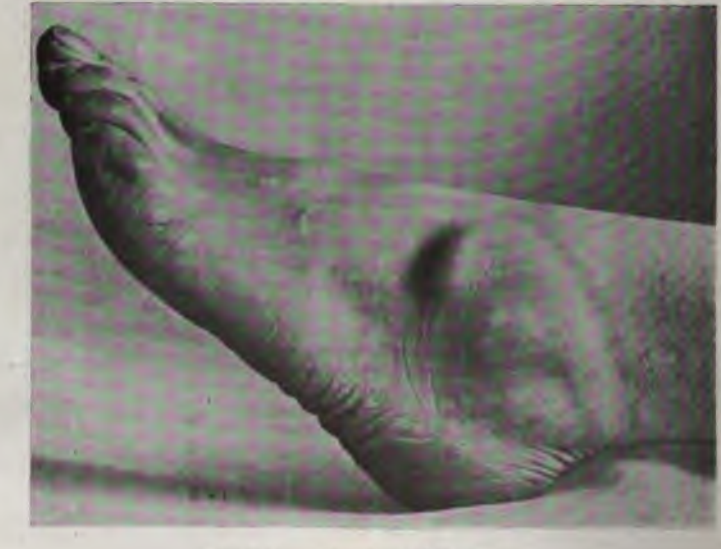


FIG. 594.—Ganglion of the tarsus.

(3) DEFORMITIES OF THE TOES.

Many an elegant shoe conceals deformities which not only offend the æsthetic taste, but which also make the life of the wearer a torture, until the patient decides to part with the toe or to permit a resection of the deformed joint.

This is especially true of the deformity, which is a product of civilization, known as **hallux valgus.** A glance at the two degrees of the deformity, as depicted in figs. 588, 591 and 592, suffices for the purposes of diagnosis. We have already referred to the secondary inflammation of the bursa which may ensue.

Hammer toe (fig. 589) is another frequent deformity. It does not depend only upon badly fitting boots, but as already stated, upon some congenital or hereditary tendency. If all the toes are affected, as in fig. 590, this assumption is all the more probable. The hammer or claw-like posture of the toes is sometimes associated with pes cavus. When this condition gives rise to pain, it is usually the result of inflammation of the bursa.

CHAPTER CX.

TUMOURS AND ULCERS OF THE FOOT.

MANY different forms of tumours and of ulcers have been observed on the foot, as on the hand, but there are very few characteristic enough to deserve mention.

(1) TUMOURS.

Ganglion, which is a tumour-like structure, should be mentioned first. It occurs on the foot just as on the hand, but is somewhat rare in the former position (fig. 594).

Chondroma of the toes is the most important of the *innocent new growths,* and its character is similar to that of chondroma of the fingers (fig. 595).

If a toe-nail is gradually raised by a tumour-like structure beneath it, a **subungual exostosis** is suggested, as already described by Dupuytren. **Fibromata** originating in the nail-bed, and growing under or close to the nail, are rare. Cutaneous horns (fig. 596) are also unusual.

Lipomata have been seen on the metatarsus and they are liable to spread between the bones and plantar aponeurosis. **Cavernous angiomata** of congenital origin also occur on the foot.

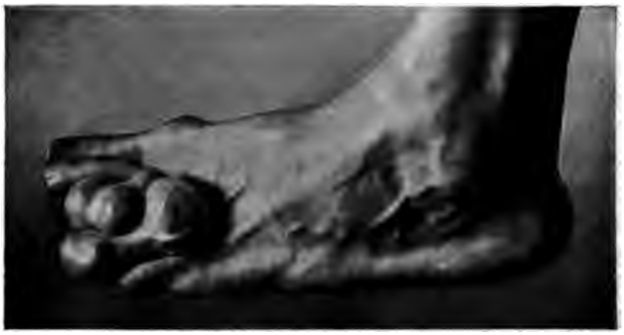


FIG. 595.—Multiple chondromata of the toes (from the surgical clinic, Berne).

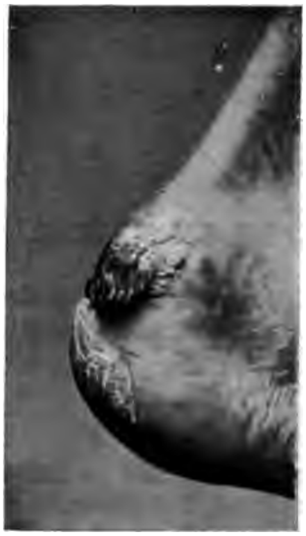


FIG. 596.—Cutaneous horn on heel.

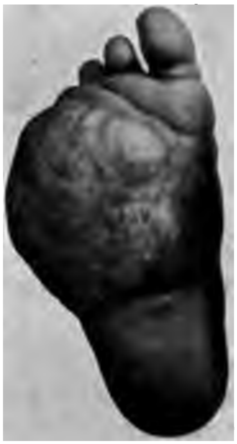


FIG. 597.—Cavernous angioma of foot.

Although these are histologically innocent, they invade various tissues such as skin, muscle and tendons, and they may lead to profound disturbances therein. Their granular surface, and their translucent bluish-red colour, and their emptying on pressure are signs which render them immediately recognizable. Fig. 597 is a typical illustration of the appearance of a cavernous angioma in general.

The most characteristic of *malignant growths* is **melano-sarcoma of the skin,** the typical features of which are seen in figs. 598 and 599. Sometimes they look rather reddish, and the true pigmentation is not recognized until they are examined microscopically. The **os calcis** is occasionally the seat of **sarcoma,** which is the most frequent of the deeper growths in this region.

(2) ULCERS.

In addition to the well-known triad of ulcers—tubercular, syphilitic and malignant—the foot presents frequent examples of "perforating ulcer" and of circumscribed gangrene of the skin. The nature of the ulcer can generally be diagnosed from its situation. In the *dorsum* they are usually tubercular (fig. 513) or syphilitic (fig. 519), rarely malignant (fig. 517). The recognition of syphilis and of tubercle has been dealt with in Chapter CIII. A malignant ulcer is differentiated from both of these by its papillomatous appearance, or by its hard edge and base. If we find a discoloration of the skin, either circular or map-like in shape, towards the toes, associated with loss of sensation in the affected area and with neuralgic pains, we must diagnose commencing gangrene and should examine for arterio-sclerosis, diabetes, or nephritis. Alcohol and syphilis may be indirect causes, if the patient is young. The gangrene becomes quite definite in the course of two or three weeks, the area involved becomes black and sloughs away from the healthy skin. The condition of the arteries and of the general circulation determines whether the process ceases with this circumscribed destruction, or whether it is merely the prelude to an ascending gangrene.

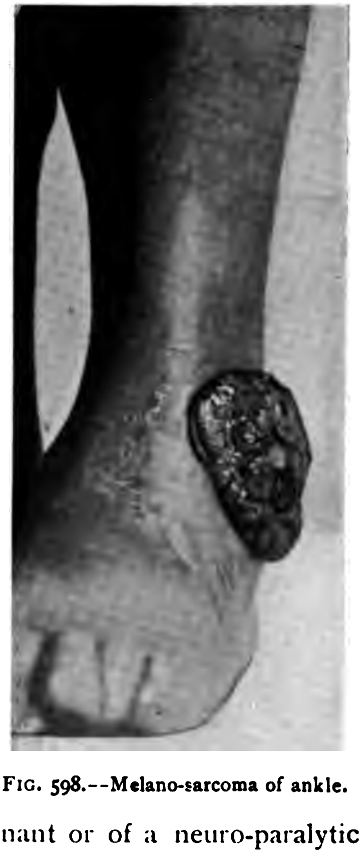


FIG. 598.—Melano-sarcoma of ankle.

An ulcer on the sole is either malignant or of a neuro-paralytic nature. The latter **(perforating ulcer)** is diagnosed from its situation on parts especially subjected to pressure, such as the heel, the ball of the great or little toe, by its slight local painfulness and its associated disturbed sensation and anæsthesia, often combined with radiating pains. These pains are not accidental accompaniments, but are indicative of the cause of the malady, i.e., of a neuritis. The margin of the ulcer is formed of thickened epithelium, and the central necrosis may extend to the tendons and bones. Attacks of phlegmonous inflammation around the ulcer are very characteristic of the condition.

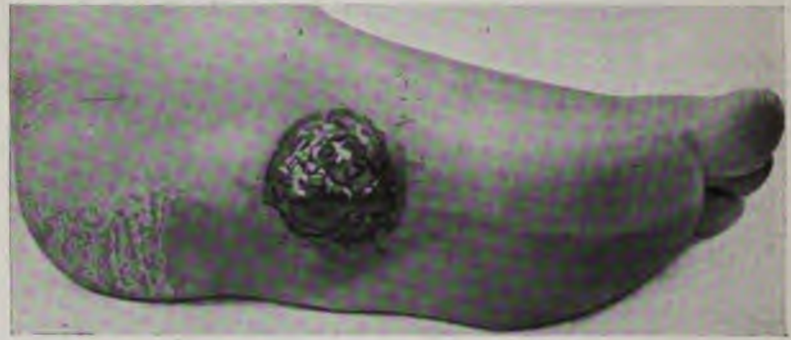


FIG. 599.—Melano-sarcoma of sole of foot.

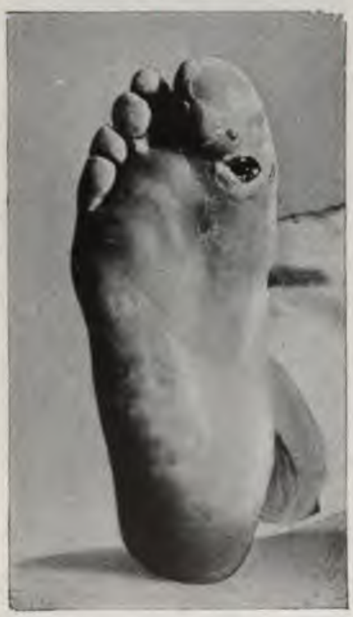


FIG. 600.—Perforating ulcer due to alcoholic neuritis.

Sometimes an injury to a peripheral nerve accounts for the perforating ulcer, but it is more frequently the result of the nerve disturbances which follow on spina bifida, or of some disease of the spinal cord or cerebral system, such as syringomyelia, tabes dorsalis or general paralysis. Alcoholic neuritis is, however, the most frequent cause.

Ulcers due to circumscribed gangrene in arteriosclerotic and diabetic subjects, as noted above, must not be confused with perforating ulcer. The former occur usually on the dorsum of the foot and toes, and are attended by severe neuralgic pains. Of course, it is quite possible that a genuine perforating ulcer may also develop in these patients.

If an ulcer forms on a part of the sole not subject to pressure it may be an **epithelioma,** which is, however, rare.

Finally, reference must be made to the ulcerative processes in the vicinity of the *toe-nails.* **Ingrowing toe-nail** is so familiar that it hardly requires mention from the diagnostic aspect, were it not that **syphilitic** and **tubercular** ulcers also occur at the same spot. The latter is sometimes termed **onychia maligna.** Syphilis is diagnosed from the history, Wassermann reaction, and the results of specific treatment. Tubercle is diagnosed if the treatment for ingrowing toe-nail and for syphilis fail to cure the ulcer; but it is a better plan to remove a small piece of the margin for the purpose of histological examination.

CHAPTER CXI.

SOME DIAGNOSTIC PROBLEMS CONNECTED WITH THE MILITARY SURGERY OF THE LIMBS.

THE War has brought into prominence many diagnostic problems connected with the surgery of the extremities, which possessed but little interest in the times of peace. Some of these points have already been referred to, but there are others which have not yet found any appropriate opportunity for discussion. It will, therefore, be convenient to utilize this final chapter for this purpose, treating the upper and lower limbs together.

These problems concern injuries to bones, joints, blood-vessels, and nerves, the demonstration and localization of foreign bodies, and the more important inflammatory complications of wounds.

(1) The Evidences of Injury to Bone.

Very few words suffice for this subject. The pain on movement is so intense, and the false mobility is so much more definite than in the case of fractures in civil practice, that there is scarcely any room for doubt. But if only one of the bones at the distal end of the leg or forearm is involved, and the damage is not serious, a skiagram will be required to make a correct diagnosis, because it is not permissible to palpate the seat of the fracture as in the case of simple fractures, owing to the great risk of sepsis. But no harm can ensue from this delay because if this doubtful condition is the wounded man's only injury, he can forthwith be conveyed to a base hospital provided with an X-ray installation. If we have diagnosed a fracture due to gunshot wound, it follows that the fracture is comminuted, because this is the condition in nearly all gunshot fractures. As far as treatment in the advanced line is concerned, it matters not whether the splinters are large or small or what their position is. The search for splinters, or the attempt to extract them has no place in modern military surgery. Although these procedures may testify to the well-intentioned zeal of the surgeon, they can only do harm. But if there be pressure on nerve trunks or on large vessels, it will be necessary to deal with these splinters early, and in these circumstances these fragments of bone are of diagnostic interest.

(2) The Evidences of Injury to Joints.

It is very important to ascertain as soon as possible whether a joint is involved in an injury, because wounds of a joint demand special care and a position of repose. It is easy enough to recognize an injury to a joint, if it is directly exposed, if the bones forming it are shattered, if the joint cavity is filled with blood, or if it lies in the direct line of the wounds of entry and exit. But the diagnosis is difficult when the joint appears to be normal and its movements free, and there is only a wound of entry which may not be absolutely in the joint line. It may be possible to make a diagnosis by considering a skiagram in relation to the wound of entry, but if this does not help the patient must be watched carefully, and if he has to be removed it is imperative to secure the joint safely with splints. If we have the opportunity of watching the case, we may probably see the joint swell in the course of a few days, and a rise in temperature. If the general and local rise in temperature is only trifling and the pain is slight, there may be nothing more than an aseptic effusion. The more severe the local and general symptoms the more likely is it that there is an infection, and therewith active treatment is required. But the possibility of an aseptic effusion must always be kept in mind, so as to avoid the risk of introducing sepsis into an aseptic joint, by hasty interference. An exploratory puncture is only justifiable in circumstances of perfect asepsis, and this is not always attainable under military conditions. If the joint is open and suppurating, a bacteriological investigation is of interest, because a pure staphylococcic or streptococcic infection is less serious than a mixed infection with the *Bacillus pyocyaneus* which is a very ready intruder. No cultures need be made for the detection of this organism, because its presence is revealed by the colour and odour of the pus. If a suppurating joint refuses to heal, the delay may be due to foreign bodies—bullets, fragments of uniform—loose pieces of bone, burrowing pockets of pus which have not been laid open adequately. This last condition occurs frequently in the knee-joint, where pockets run from the popliteal space into the leg and thigh. Patience and diagnostic acumen will save many a limb from amputation. It is most important not to rely upon the statements of the wounded man, for details of his surgical history at the advanced dressing stations. Each case must be examined by X-rays, and suppurating pockets must be opened in order to render the diagnosis clear.

(3) The Diagnosis of Injuries of the Larger Blood Vessels.

We shall not discuss primary hæmorrhage in this connection. As a rule this has already been arrested or has ceased spontaneously before the patient is brought to the dressing-station. At any rate, there is never active hæmorrhage when he is brought into hospital. We are most concerned here with the wounds of blood-vessels which are overlooked at first, but which lead subsequently to hæmorrhages or to aneurisms. We should always think of this possibility, when the direction of the bullet crosses or coincides with the course of a large vessel. We may be quite certain of this injury, if the pulse wave below is arrested, and if coldness and pallor distally to the wound herald the approach of gangrene. In other cases a *secondary hæmorrhage* indicates that a blood-vessel has been wounded. The wound, which is otherwise healthy, begins to trickle, and though it does not, at first, appear to be at all serious, the oozing persists despite a pressure bandage. At other times, the premonitory hæmorrhage is sharper, but it ceases spontaneously. Sometimes, however, the hæmorrhage sets in with such severity, that it would soon be fatal, unless skilled assistance were available. These incidents usually occur in the second or third week, even in non-infected wounds, but they are, of course, more frequently due to septic softening of protective thrombi. The moral is obvious: every wound of a large vessel requires ligature in appropriate position, above and below.

The development of *aneurism* is sometimes the first indication of a blood-vessel having been wounded. In thinking of most so-called aneurisms, which occur after gunshot wounds, we must discard our conception of the true aneurism of civil practice. In most cases, the condition is one of *false aneurism*, i.e., a blood cavity close to the lacerated vessel, with an aperture leading to the exit or entry wound, more or less blocked up by clot or displaced muscular tissue. This cavity contains liquid blood, clot and the lacerated or torn artery or vein. If the aneurism continues to develop towards the surface, without any further hæmorrhage, it gradually acquires a firm capsule of connective tissue, and the blood cavity itself, becoming more and more even, eventually becomes a sacular appendix of the injured vessel. The diagnosis is very easy if this sacular development manifests itself by pulsation or a palpable or audible murmur. There is difficulty however, if nothing but a gradually increasing firm swelling develops in the muscular tissue of the thigh, the calf or under the pectoralis major, which suggests a deep phlegmon, more especially as there is a local rise in temperature. But the inexperienced should be saved from the error of cutting into it, in mistake for a phlegmon, by the absence of any *general* rise in temperature, and possibly by hearing a murmur on auscultation. The experienced surgeon, however, knows that this process conceals a deep aneurism, and he will arrange his treatment accordingly. The differentiation between a purely arterial and an arterio-venous aneurism is of special importance from the point of view of prognosis, because the latter has a greater tendency to cure after the use of simple pressure or even spontaneously. The distinction can be made with some approach to accuracy by the character of the bruit; in an arterial aneurism, it is intermittent and synchronous with the pulse, in an arterio-venous aneurism it is continuous, but rises and falls in intensity. An aneurism may occasionally develop very late indeed, even after months; but in such a case it would be very suspicious of a true traumatic aneurism, because a blood extravasation into what was previously an extra-vascular space, would not be so long in declaring itself.

(4) The Diagnosis of Nerve Injuries.

We will assume that the injury of the nerve trunk has been duly recognized and localized, and has not been overlooked in the abundance of other injuries, as happens so frequently. For this purpose it is only necessary to bear in mind the motor and sensory nerve areas, which can be referred to in plates 1 to 3. The questions which the surgeon puts to the neurologist concern the nature of the nerve injury and its prognosis; but the surgeon ought to be able to answer these questions himself, if an answer is at all possible.

If we expose the injured nerve, we may find no gross anatomical change (i.e., concussion or shock), or an effusion of blood and thick infiltration, without any interference in the continuity of the nerve (contusion), or, finally, partial or complete rupture. If the injury is of some weeks' standing, we find the nerve or nerve trunks (brachial plexus !) embedded in an indurated mass, and it may be very difficult, indeed, to dissect out the individual nerves.

How far can we proceed with the clinical diagnosis? We begin by estimating the voluntary motor and sensory paralysis. If there be a pronounced difference between the two, we are justified in assuming that the nerve has not been completely divided; but one must not forget, in mapping out areas of sensation, that there is a good deal of overlapping of adjacent nerves.

We then test the electrical conductivity of the nerve. If this still persists, it is obvious that the nerve has not been completely divided; but if the conductivity is lost, it by no means follows that the nerve *is* divided. As a matter of fact, experience shows that very few gunshot wounds cause total severance of a nerve. But the probability of this having happened increases the longer the loss of conductivity persists, and if severe degeneration changes in the muscle set in (i.e., reaction of degeneration, total absence of all electrical reactions). But even this does not necessarily imply anatomical division of the nerve; it merely means that there is some serious cause preventing the restoration of nerve function (pressure by callus or firm induration, splinters of bone or of projectile). In many cases there is a return of voluntary motor power and electrical conductivity in a few weeks. But if there is no sign of any return of function after four to six weeks, it means that the damage is sufficiently serious to necessitate operative treatment for its repair.

We have been assuming hitherto that the paralysis follows the wound forthwith. Sometimes, however, the paralytic symptoms come on gradually, and often in association with sensory irritative symptoms, i.e., neuralgia. The diagnosis is easy in such cases, for the symptoms are due to callus growing around the nerve, to neuritis or to aneurism. If circumstances point to *compression by callus*, the treatment in civil practice consists of immediate operation. In military practice, however, it is better not to hurry the operation, but to wait until the gunshot wound has closed, in order to secure better asepsis. Infection may be lurking, not only in the injured bone, but also in the indurations in the track of the projectile through the soft parts. The French aptly term this condition "microbisme latent." If neuralgia supervenes after a gunshot wound, which has not injured the bone, it can only be due to *neuritis*. But we should not make this diagnosis until we have excluded the existence of an aneurism in the vicinity of the painful nerve trunk. This is not at all difficult, if one bears in mind that aneurism is a cause of neuralgia. The term neuritis is often used very indefinitely. Sometimes the operation reveals the nerve trunk converted into a cord nearly as thick as a lead pencil. At other times the neuralgic pains appear to be due to the imbedding of the nerve in an unusually firm and thick fibrous tissue, of the same character as is sometimes left when a gunshot wound of the soft tissues heals without suppuration. The condition is analogous to compression by callus.

(5) The Evidence of Presence of Foreign Bodies.

The search for bullets was the main occupation, if not the pastime, of the older military surgeons. All the complications of the wound were ascribed to foreign bodies. They were right to a certain extent, taking into consideration the infantry bullets of 15 millimetres and more calibre, which were used fifty years ago. To-day, however, the position is absolutely different. Not only can we demonstrate the presence of every bullet, but we can also determine their position. This might be thought to lead to much operative activity. But, as we know, that modern bullets in the body are only injurious under certain conditions, our zeal for extracting them is checked, and our treatment is directed along certain definite lines. Sometimes the wounded man refuses to have the bullet extracted, when *we* wish to do so; but more frequently he compels *us* to extract a bullet which we consider harmless, because he wants to hang it on his watch-chain as a trophy, or has some morbid fancy about being poisoned by lead or copper. It has, indeed, been proved that absorption of lead can take place, but we do not know how often this occurs; but, at any rate, the risk of its occurrence is not great enough to justify an extraction which may be a dangerous procedure. I have seen the French copper-bronze bullet lying in the wound after weeks, and even months, as bright as when it left the rifle. There is, of course, no danger in the nickel or steel of the covering of the bullet. In considering the question of removal, a correct localization of the projectile is most essential, because this knowledge enables us to decide whether it can be extracted with a minimum amount of damage or not.

I make no reference to such adventitious aids as bullet-probes, acupuncture and telephone probes, because bullets ought only to be extracted in a properly equipped hospital, provided with the indispensable X-ray apparatus. The surgeon at the advanced dressing-station ought to concern himself about a bullet, as little as the civil practitioner who promptly sends a case of bullet wound into hospital. When the newspaper reporter writes that the " bullet was not found," and looks upon this as something of a surgical opprobrium, he merely exposes his own limitations of knowledge.

By means of X-ray examination we seek to establish the presence of the projectile, and to determine its position and character. We must consider whether a *screen-examination* is adequate, or whether we must resort to an *X-ray plate.* The former has the advantage of rapidity, and employs but little material. It is useful for the purpose of deciding whether there is a large piece of metallic foreign body in the tissues, and it gives its approximate position. But the screen usually fails to reveal fine splinters of shell, or even larger pieces of projectile when they are situated in the thick parts of the body. A screen examination can, therefore, only be looked upon, as giving some information preliminary to taking a plate, and our main reliance must be placed on the latter, despite all the modern radioscopic aids to operating, including the X-ray operating table. I have seen a very experienced investigator making fruitless attempts to determine the depth of a projectile in the shoulder both before and during the operation, whereas a stereoscopic plate, taken as described below, gave the necessary information in a very short time, with mathematical accuracy.

The examination by plates must be conducted methodically, and with scrupulous precision.

A reliable localization requires the following impressions:—

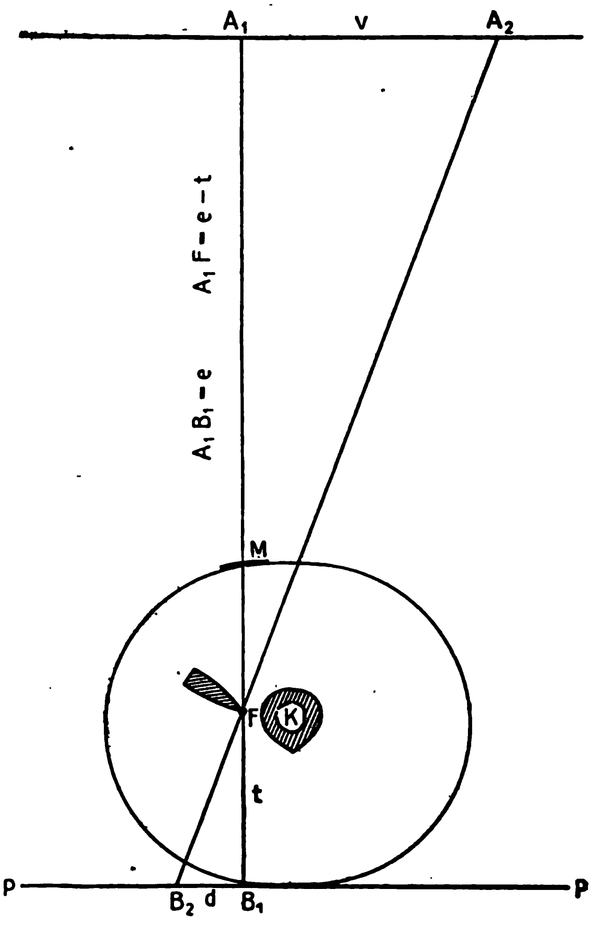
(*a*) *Two impressions in two planes at right angles to one another;* one of these should, if possible, correspond to the plane of the field of operation.

In certain exceptional cases it is impossible to obtain two impressions at right angles to one another. In the case of the hip or pelvis, for example, the antero-posterior impression is easily taken, but the profile impression can only be obtained, when the projectile is not too small and the apparatus is very good, by means of two intensifying screens. The other parts of the body, abdomen, thorax, present no special difficulties in this respect, nor does the arm, if it can be abducted. As already stated in connection with fractures, the plate is laid upon the shoulder, the arm is abducted in the horizontal and the light is directed from the axilla.

It is important that the skiagram be taken so that the bullet lies as much as possible in the direct line of the anti-cathode, otherwise errors will occur in the marking. If the plate has not been taken with this object kept sufficiently in view, it must be re-taken in an improved position.

The two skiagrams taken in this way, usually give enough information for the extraction of the projectile, if it lies in the proximity of a characteristic part of a bone. If it is in the soft tissues of the extremities in a position wherein the shape of the bone does not aid in the localization, it is necessary to ascertain its relation to the surface by means of wire markers, whose position on the skin is marked, or by means of a special wire triangulation. A screen examination renders some assistance in arriving at a true representation of the position of the foreign body, in these circumstances, especially if the observer is unable to co-ordinate the two pictures so as to arrive at the correct position. But there are cases wherein this is not sufficient, and we must resort to the following method, which gives uniquely accurate results:—

(*b*) *A stereoscopic impression for the purpose of determining the depth,* *by means of parallactic displacement.* The mathematical principle underlying this method, first employed by Perthes, is the basis of all methods of really accurate localization, by whatever name they may be known. The mathematical calculation is extremely simple, but it has been still further facilitated by means of apparatus devised by Fürstenau, Schulz, Canovas, Mackenzie Davidson, Weski and others. We will, however, assume that the hospital is not provided with this special apparatus, but merely possesses an ordinary X-ray outfit, and has some mathematical knowledge available.



F = Apex of projectile.
M = Metallic marker in line of anticathode.
K = Bone.
A_1 and A_2 = the two positions of anticathode.
B_1 and B_2 = the corresponding shadows of point F.
P—P = Plate.
A_1A_2 = Displacement of Röntgen tube, i.e., v.
B_1B_2 = Distance between the two pictures, i.e., d.
B_1F = Distance of foreign body from plate, i.e., t.
A_1F = Distance of foreign body from anti-cathode, i.e., e—t.

$$t : d = e - t : v.$$
$$v\,t = de - dt.$$
$$t = \frac{de}{d + v}.$$

FIG. 601.—Determination of depth of foreign body by means parallactic displacement.

Our task is to determine the distance of the foreign body, or rather a given point on it (F) from the surface, or from a plane resting thereon (X-ray plate, p-p). This plane should as far as practicable be the one in which the operation is to take place, if the plate can lie in this position. In order to control our calculation, and at the same time to facilitate accessibility from the opposite side, we fix a metallic marker (M) of characteristic shape, e.g., a triangle, on the skin in the line of the anti-cathode, and in the projection of the foreign body, the position of which has been approximately obtained from our previous investigations. A wire indicator, e.g., of □ shape, is fixed on the plate side of the part of the body concerned, in the same projection, in order to aid localization during the operation. We now take two impressions upon the same plate, with the same time exposure. The former is taken accurately in the line of the lead marker and the direction of the foreign body, and for the latter the X-ray tube is moved a definite distance, e.g., 10 c.m. (6.5 cm. with Fürstenau's apparatus). These two impressions can be taken simultaneously with two separate anti-cathodes, by means of the tubes recently put on the market. The plate will therefore present two pictures of the foreign body, the bone and the lead marker, and one picture of the wire indicator lying on the plate, the position of which is marked on the skin with caustic. The distance from the opposite side, between identical points of these pictures enables us to calculate their distance from the plate. The accompanying diagram (fig. 601) shows how the results are obtained. It is however indispensable that, in the one impression, the direction of the rays must fall absolutely perpendicularly through the foreign body, on to the plate. In practice this will not always be the case;

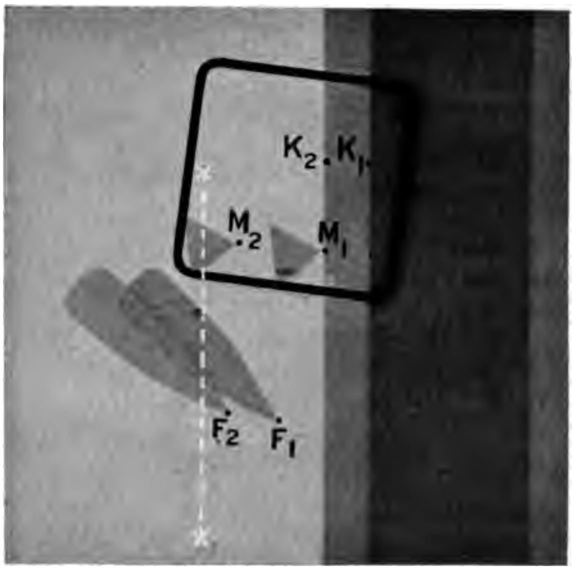


FIG. 602.—Skiagram corresponding to fig. 601. Determination of position of bullet in thigh.

but this does not affect the mathematical relationship of the sizes which concern us, because the formula applies to any position of the tube.

According to this formula, it is possible to determine the distance of the plate from the various points which we find in the skiagram represented in fig. 602. We simply substitute the measured distances F_1, F_2, M_1, M_2, K_1, K_2, in series, for d in the formula. As we can obviously measure the distance A_1 M directly, we obtain a control for the accuracy of our calculation. The distance of the foreign body from the marker M is obtained by subtraction (B_1 M—t). We decide upon the side for extraction according to whether the distance of the foreign body is greater from the plate or from the marker. If we also determine the depth of the *base* of the bullet, we can ascertain whether this lies parallel with the plane of the plate, or whether it is more or less inclined to it.

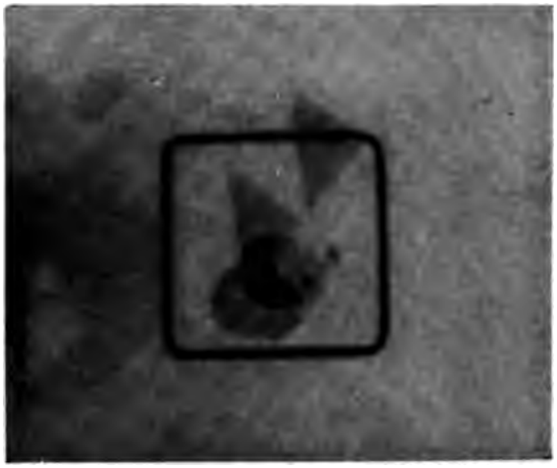


FIG. 603.—Bullet in skull at posterior end of corpus callosum. An example of precise delineation.

F_1 and F_2, M_1 and M_2, K_1 and K_2 are identical points of the bullet, the marker M and the bone. If we represent the respective distances by the formula $\frac{d\,e}{d + v}$, the vertical distance of the depth is obtained, *i.e.*, the vertical amount of interval from the plate. In the case illustrated $e = 60$ cm. and $v = 6{\cdot}5$ cm.

As F_1 and $F_2 = 0{\cdot}7$ cm., therefore the interval between the bullet and the plate $= \frac{0{\cdot}7 \times 60}{0{\cdot}7 \times 6{\cdot}5} = 5{\cdot}8$ cm.

As $M_1\,M_2 = 1{\cdot}3$ cm., the interval between the marker and the plate = 10 cm., etc.

The agreement between the projection of the marker and of the bullet is in this case sufficient for the purpose of estimating the depth. But, on the other hand, the wire triangle lying on the plate side of the thigh which should delimit the field of operation, is situated some 3·5 cm. too much towards the knee, having regard to the apex of the bullet. The incision must, therefore, be planned more towards the hip, and at the same time about 1 cm. further away from the bone, i.e., in the direction X—X. The precise horizontal intervals of the

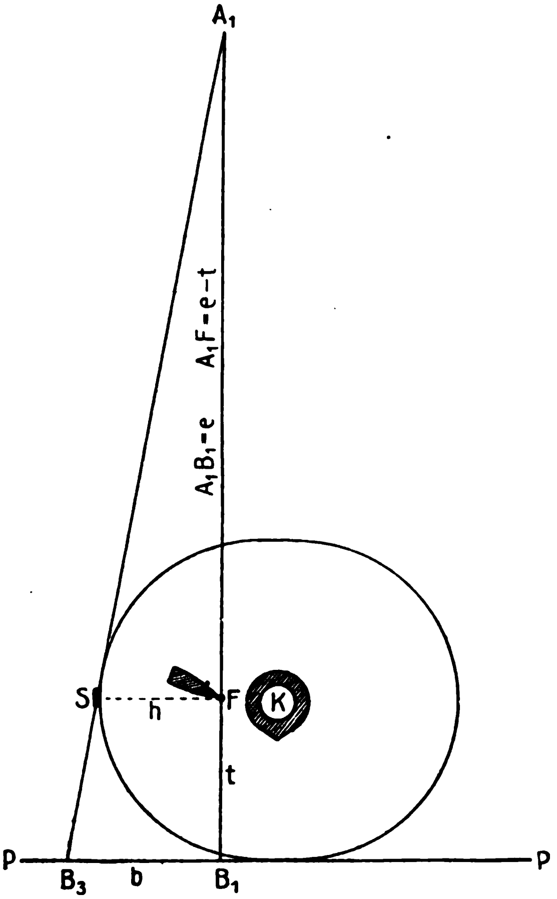


FIG. 604.

separate points of the picture cannot be read off the skiagram forthwith, but their values must be found from the formula $h = b\left(1 - \frac{t}{e}\right)$ deduced from fig. 604. If, for example, the apex of the bullet on the skiagram appears to be 2·6 cm. distant from the lead marker M, this distance is necessarily less on vertical projection of both points. If we substitute the difference in depth between MB_1 and FB_1 for t in the above example (10—5·8 = 4·2) we obtain as the real horizontal distance h = 2·6 $\left(\frac{60-4\cdot2}{60}\right)$ = 2·4 cm.

On the other hand, the horizontal distance between the foreign body and the wire triangle corresponds directly with the distance measured on the plate, when the anti-cathode is vertically placed over the foreign body—a position whose importance must be emphasized. If the line of the anti-cathode deviates from that of the foreign body to the exert of V_1 cm. the correction can be made from the easily deduced formula $h = b \pm \frac{t + v_1}{e - t}$. The *minus* sign is employed if the anti-cathode deviates towards the same side as the wire triangle, and the *plus* sign, if the direction is reverse. In our example the deviation is 2·4 cm.; the horizontal distance from the apex of the bullet to the middle of the quadrilateral figure is therefore not 3·5 cm., as measured on the plate but 3·3 cm.—a considerable difference in the range of other sources of error.

It is sometimes important to know the horizontal distance of the depth (i.e., the distance FS in fig. 604) as well as the vertical. This is done most easily by taking another skiagram on a new plate, and placing a lead marker S on the skin, at the spot corresponding to the interval t previously found. The following conditions thus obtain :—

S = side marker.
B_3 = shadow thereof.
F S = h – the horizontal depth distance required.
$B_1 B_3 = b$ = shadow interval between apex of bullet and marker S
(e — t) : e = h : b.

$$h = \frac{b\,(e - t)}{e} = b\left(1 - \frac{t}{e}\right).$$

In some cases it is important to determine the comparative depths of certain points on the bone and the bullet. This helps us to decide whether the projectile lies in front of, behind, or in the humerus, or head of the femur.

For example : A bullet has penetrated in the region of the great trochanter, and the skiagraphic localization shows it to be in the vicinity of the neck of the femur. No sign of bone lesion. The X-ray apparatus employed is of an old design. The parallactic displacement gives a depth of 8½ cm. from the inguinal region, and the same distance from behind, that is to say, the same as the depth of the neck of the femur. The operative incision was made from the front. No bullet was found in the neck of the femur at the depth of 8 cm. The neck was opened by chisel, and the undamaged pointed bullet was found lying under the cortex of the bone, exactly at a depth of 8½ cm.

A correct appreciation of the questions involved in these simple problems will facilitate the solution of less simple ones, and those who are aware of the tediousness of searching for a badly localized bullet, will not venture upon an exploration without the guidance of an accurate skiagram—unless, of course, the bullet is accessible to direct palpation.

The nature of the projectile is often obvious at the first glance—rifle, revolver or shrapnel bullet, or piece of shell. Sometimes a certain amount of experience is required to identify it, and it is often impossible to distinguish a misshapen shrapnel bullet from a piece of shell. But apart from these irregularities, the bullets of hand weapons present their own interesting problems. Revolver and Mauser bullets are recognizable in profile by their slight length. Entire rifle bullets—oval or pointed—possess a characteristic appearance, at any rate in one of the impressions. Larger irregular jagged shadows, with numerous smaller splashes amid the fragments of a crushed bone—usually a diaphysis—show that a sheathed bullet has broken the bone at a comparatively short distance, and has in the process become ripped up itself. The presence of bits of lead in a skiagram is, therefore, no evidence of the use of "dum-dum bullets." Even if, after firing at short range, without the occurrence of a fracture small fragments of the lead of the bullet be found scattered about, this may be due to impact of the bullet against some hard object in proximity with the wounded man, e.g., the barrel of his gun. I once saw five soldiers with fragments of lead and bullet sheath in their feet and calves, resulting from the impact of a pointed bullet against a stone slab in the group. There are some cases, however, wherein the presence of numerous bits of lead, depends upon the constitution of the bullet. In these cases there is usually a large wound cavity, not sufficiently accounted for, by the bone damage. But even then, our opinion must be guarded, because rotating bullets, and those which rotate in the wound itself can produce equally extensive damage.

We cannot go into details as to the information given by the shape of the entry and exit wounds, by the bullet track, and by the position of the axis of the bullet. This is exclusively a matter of pure military surgery.

Hitherto we have limited the discussion to metallic foreign bodies; but it is easy to detect pieces of stone and glass in a skiagram, whereas the presence of bits of cloth, leather and wood can only be surmised in cases of infection, when the suppuration is persistent. This leads us on to the last section of this chapter.

(6) The Diagnosis of Infections of Gunshot Wounds of the Limbs.

Although the bacteria which lurk around wounds of battle are the same as those of civil conditions, nevertheless the special circumstances of warfare and the limitations of surgical treatment outside hospital wards, leave their stamp upon these wounds, and present special diagnostic problems to the surgeon.

Less than half a century ago the ordinary infections of the battlefield were intensified by unnecessary surgical interference, and by the absence of the most elementary cleanliness. To-day, if we are unable to prevent most of the primary infections, at any rate we do not ourselves inflict any secondary infection on the wounded man.

The most important infections are divisible into four groups, each of which possesses its own diagnostic interest.

(*a*) *Ordinary Suppuration.*

The staphylococcus and streptococcus play the chief rôle here, but the *Bacillus coli* and *B. pyocyaneus* are frequently associated. Every wound of battle, just as every accidental wound must be regarded as infected from the bacteriological standpoint, though the infection does not become active in every wound. Even if this does occur, it varies much in degree. A simple wound may merely exhibit a harmless, superficial inflammation, whereas a dirty, irregular, sinuous wound caused by a piece of shell may lead to a deep phlegmon with the subsequent formation of pockets of pus in all directions. The diagnosis of these conditions is a matter of general surgery. It is only necessary to remark that obstinate and profuse suppuration despite free incision and drainage of pockets, and especially a firm hold of the *B. pyocyaneus* in the wound, almost always indicates a foreign body; not merely a bullet but more frequently a sequestrum or a piece of necrosed soft tissue (tendon, fascia), and especially a shred of cloth or a piece of leather which has penetrated with the bullet. Pyocyaneus pus is not always bluish-green and does not always smell so powerfully as in the classical example. It often resembles the pus of old tubercular burrowing abscesses.

It is not necessary to dwell upon the diagnosis of generalization of the infection, in the form of pyæmia. This generalization has now become an event of very much less frequency than previously, and this constitutes one of the great advances of modern military surgery.

Sometimes the patient's temperature remains persistently high, although there is nothing in the wound to account for it. The surgeon may search in vain for foreign bodies, burrows of pus, phlebitis and general infection, and all the time may be overlooking *typhoid fever*, which was in the incubation period when the patient sustained his wound, or which originated after the infliction of the wound.

(*b*) *Erysipelas.*

Although we do not assign any specific character to the streptococcus of erysipelas, the disease must be regarded as a special form of infection—not indeed peculiar to the battlefield, but almost always the consequence of too much surgical interference. Probing about, necessarily and especially unnecessarily, into a suppurating wound is often followed by a sharp rise of temperature to 102° F. or even 104° F. with headache and general malaise, and then, when the dressing is changed, a commencing erysipelas is often detected. In the present war, cases of erysipelas are rare—an evidence of repression of surgical zeal in the treatment of wounds.

(c) *Infections associated with Gas Formation.*

The surgical literature of the present war would suggest that "gas phlegmosis" constitute a new form of infection; but observant surgeons in large civil practice are quite familiar with this condition. The earliest stage of this infection can be recognized in war surgery, if wounds are carefully observed.

These gas infections are not due to any one bacteriological cause, nor are the resulting clinical pictures alike. Fränkel's gas-forming bacillus is apparently the most frequent causal organism, generally associated with the usual organisms of suppuration. Members of the group of organisms to which the bacillus of malignant œdema belongs, are less frequently found.

The wounds affected are dirty, irregular and sinuous, both in civil or military practice. They may be contaminated by street refuse (compound fractures), garden soil (shell wounds); fæces (with intestinal infection) or putrid meat. I have seen a case resulting from the bite of a lion, after the animal had been fed. The clinical pictures are as follows:—

The process may be limited to a few bubbles of gas, which come up now and again from the depths of the wound, with some evil-smelling pus. The formation of gas occurs in the cavity of the wound rather than in the tissues, and it ceases spontaneously after a foreign body or a piece of dead tissue has been extruded. These are circumscribed gas-abscesses, not gas-phlegmons—and they remain circumscribed.

In other cases, a thick œdematous swelling of the skin and subcutaneous cellular tissue appears around the wound, on the first or second day. Its peculiar brownish, greyish-brown or yellowish-brown aspect is very significant to the experienced observer. French authors term it "œdème bronzé" (see plate 5). It is distinguished from erysipelas by the extensive depth of the swelling, and by the distinctive hue. The swelling spreads just like erysipelas, with more or less rapidity; it can sometimes be seen to extend hour by hour. It may reach from one of the limbs right to the trunk, and involves the tissues more or less deeply.

The tissue necrosis and the development of gas, though possibly of different intensity, follow rapidly upon the bronze coloured swelling. There is no sharp demarcation between the cases wherein phlegmonous processes predominate, and the third type which is characterized by complete gangrene of a large portion of a limb. There are gradual transitions both in severity, and in rapidity from the mild cases to those with erysipelatoid characters, with discoloration and gas-formation, and fulminating gangrene of an entire extremity. The sooner the practitioner—in peace and in war—recognizes the malignancy which lurks in the colour and the extension of the phlegmon, and in the gas-formation, the greater is the prospect of rescuing the life of the patient and even saving the affected limb.

(*d*) *Tetanus Infection.*

We have already discussed the diagnosis of Tetanus, in connection with head tetanus (Chap. 13). It is only necessary to make one further remark here. The beginning of the spasm is liable to be overlooked, even in hospital, because the "difficulty in swallowing" of which the wounded man complains at the morning visit, is so often assumed to be due to sore throat.

It is well known that tetanus more often supervenes after wounds inflicted by soil-contaminated pieces of shell, than after bullet wounds. But the difference is not so great as to justify less apprehension of its onset, after the latter wounds. We sometimes see in civil practice that the slightest skin wound suffices for an attack of the tetanus bacillus. It is noteworthy that infections of wounds in war including gas phlegmon and tetanus are particularly prevalent in the lower extremities.

INDEX.

A

Abdomen, surgical diseases of, 251
—, injuries of, 260
— —, with external wound, 268
— —, without external wound, 260
Abdominal cavity, acute inflammation in, 270-293
— —, effusion of bile into, in injuries of liver, 263
— —, entrance of gas into, in ruptured intestine, 261
— — — — — — — —, in perforation of stomach, 331
— —, tumours in, demonstration of, 304-308
— — — —, by exploratory puncture, 308
— hernia, epigastric, 393, 394
— injuries, gunshot, 268
— integument, abscess of, 284, 392
— —, actinomycosis of, 400
— —, fibroma of, 398, 399
— —, tenderness of, in peritonitis, 274, 275
— —, tumours of, 304, 391-400
— — —, atypical positions, 399
— — — in inguinal region, 397
— — — lumbar region, 398
— — —, umbilical region, 394
— — — upper abdomen, 392
— lipoma, subcutaneous, 393, 399
— muscle, tuberculosis of, 399
— nævi, sarcomatosis of, 232, 399
— rigidity, reflex after intestinal injuries, 262
— — — — —, in peritonitis, 274, 275, 291
— sinuses, 401
— — in groin, 402
— — at umbilicus, 401
— tumours caused by inflammatory changes, 305
— — causing intestinal obstruction, 305
— —, consistence of, 307
— —, determination of origin of those which fill up entire abdomen, 306
— —, innocence and malignancy of, 308
Abdominal tumours, movable, 306
— —, phantom, 304
— —, slightly movable and not very extensive, 306
— —, type of displacement of unpaired organs, 252
— viscera, diagram of parts felt on palpation, 255
— — displacements, 251; congenital, 251; acquired, 253
— wounds, perforating and incised, 269
Abducens nerve (6th), focal symptoms in lesion of, 43
— —, paralysis of, in fractures of base of skull, 4
Abrasion fracture of eminentia capitata humeria, 599, 600
Abscess, appendicular, 356, 357; position of, 356
— —, diagnosis from hip disease, 692
— — — —, peritonitis, 281, 282, 283
—, Bezold's, 35, 142
—, cerebellar, 34
—, epidural, 32
—, epigastric, 392, 393
—, gluteal, 552
—, inguinal, 552
—, meso-cœliac, diagnosis from peritonitis, 281
— of axilla, 224
— brain, 18
— —, diagnosis, 18, 19
— —, etiology, 20, 21
— —, symptoms, 18, 19, 20
— breast, 235, 236
— —, superficial, 236
— kidney, 471, 481
— liver, 282, 347
— lumbar region, 398, 399, 550
— lung, 208, 209
— —, diagnosis from bronchiectasis, 211
— neck, 139, 141, 550
— —, chronic, 143, 144
— —, due to caries, 549
— —, tubercular (cold), 143, 145, 148
— palate, 103
— pelvic bones, 550, 551
—, peri-articular, of foot, 772

Abscess of seminal vesicles, 458
— spleen, 282, 351
— supra-clavicular fossa, 141
— supra-symphysis pubis, 284
— temporal lobe, 33, 34
— tibia, lower end, 749
— — knee, 721
—, perineal, 552
—, peri-nephritic, tubercular, 399, 471
—, peri-proctal, 436, 458
—, pharyngeal, 104
—, posterior cervical, 142
—, prostatic, 458, 501
—, residual, in peritonitis, 272, 288, 355, 358
—, retrobulbar, 54, 65
—, retromammary, 236
—, retroperitoneal, 283, 356
—, retropharyngeal, 99, 121
—, retrotonsillar, 99
—, submental, 140
—, subphrenic, 208, 293
— —, in appendicitis, 358
— —, with pleural exudation, 297
— —, without pleural exudation, 294
—, sub-umbilical, 284, 401
—, tubercular (cold), of skull, 60, 61
— — — of spine, 143, 226, 544-553
Accessory goitre, 104
— —, true and false, 159
— thyroids, malignant growths of, 182
Acetablum, fractures of, 656
—, displacement of, in hip disease, 690, 693
Achillodynia, 773
Acne pustules of axilla, 224
Acromegaly in tumours of pituitary body, 25
Acromio-clavicular joint, injuries of, 570, 571
Actinomycosis of ileo-cæcal region, 305, 400
— jaw, 83, 84
— limbs, 604
— lungs, 208, 223
— mamma, 237
— neck, 144, 149
— —, with sinus formation, 145
— tongue, 115
Adamantinoma, 95
Adenoids of roof of pharynx, 99
Adenoma of liver, 346
—, testicle, 449
Adiposis dolorosa, 26, 185
Air-goitre, nature of, 150
Air passages, diseases of, chronic, 125
— — —, surgical, 118
Air passages, foreign bodies in, 123, 124
— —, injuries of, 123, 202
Albuminuria in diphtheria, 120
Alveolar process, hæmorrhage in fracture of upper jaw, 81
— tumours, 93
Anæsthesia dolorosa in tumours of jaw, 92
— paralysis, of upper limb, 631
Anal region, fistulæ, 436, 450-454
— — — complete and incomplete, 453
— — —, nature of, 452
— —, injuries of, 437, 438
— —, prolapse of, 436, 437
— —, tumours of, 433-435
Anarthria, diagnosis and surgical significance of, 49, 50
Aneurism, cirsoidal, of face, 68
— of aorta, 214-219
— —, penetrating through thoracic wall, 228
— carotid, 173
— fibula, 745, 751
— innominate artery, 217
— neck, 135, 172
— —, arterio-venous, 174
— —, diagnosis from other tumours of neck, 149, 173, 174
— thigh, 698
—, ophthalmic, 53
—, popliteal, 730
—, subclavian, 173, 183
Angina, diffuse and unilateral, 98
—, dysphagia in, 130
—, Ludwig's, 98, 140
Angioma of arm, 608
— cavernous, 57
— face, 68, 69
— foot, 788
— hand, 638
— longitudinal sinus, 58
— mouth cavity, 100
—, mulberry-like, of infant, 58
— orbit, 53
— sacral region, 517
— scalp, 55
— thigh, 698, 699
— thoracic wall, 229
— tongue, 113
Ankle, contusions of, 752
—, dislocations of, 759-763
—, fractures of, 752, 754-758
—, gonorrhœal effusion of, 772
—, injuries of, with deformity, 759
— —, without deformity, 752
— —, summary of, 767
—, sprain of, 752, 755

Ankle, tuberculosis of, 773, 774
Ankylosis of hip-joint, 695
— jaw, causes, 85, 86
— knee-joint, 728
— vertebral joints, 554
Anuria, 460
Aorta, aneurism of, 214-219
— —, skiagraphy in, 217
— —, diagnosis from tumour, 214, 304
Aphasia, focal diagnosis of, 49, 50
— in cerebral abscess, 33
— — cerebral pressure, 15
Appendicitis, 285, 353-360
—, chronic, 366
—, complications, 357-359
—, condition of vermiform appendix and its surroundings in early stages of, 353
—, diagnosis of, 353
— — in the intervals, 360
— —, from abdominal tumour, 305
— — — cholecystitis, 335
— — — hip disease, 692
— — — peritonitis, 285
— — — ruptured tubal pregnancy, 290
— — — urinary tuberculosis, 489
—, diagrams of its most important forms, and some diseases concerned in differential diagnosis, 286-289
—, exploratory puncture in, 360
—, history of, 285, 359
—, ileus in, 358, 376
— in hernial sac, 425
—, pregnancy and puerperal period, 292
—, larval (Ewald), 359
—, physical signs, 359, 360
—, position and extent of appendix abscess, 356, 357
—, residual abscesses and their significance, 355, 358
—, symptoms in localized peritonitis, 354
— — in early stage, 353
— — when accompanied by general peritonitis, 355, 356
— with peritoneal symptoms, 282, 355
Appendix vermiformis, abscess of, 286, 354
— —, displacement of, 253, 336
— —, its condition and surroundings in early stage of appendicitis, 353
— —, palpation of, 256
— —, position of, 356
Arm, chondroma of, 611
—, fibroma of, 611
Arm, gumma of, 609, 610, 611
—, mobility in injuries of spinal column, 521
—, muscular angioma of, 608
—, muscular tuberculosis of, 608
—, myositis ossificans of, 610
—, neuroma of, 607
—, osteoma, traumatic, of, 610
—, osteomyelitis of, 611, 612
—, posture of, in transverse lesion of spinal cord, 522
—, sarcoma of, 607, 611
—, tuberculosis of, 610, 611, 612
Artery, femoral, aneurism of, 698
—, mesenteric, embolism of, 281
—, ophthalmic, aneurism of, 53
Arthritis, acute, of elbow, 604
—, deformans, 626, 627
— of foot, 771
— hip, 694, 695, 696
— jaw, 84
— —, ankylosing, 85
— shoulder, 584
— vertebral joints, causing chronic deformity, 554
— wrist, 626
— —, tubercular, 625, 626, 627
Articulation, sacro-iliac, sprain of, 670
— — —, fissure in neighbourhood of, 670
Ascites, chylous, in old umbilical herniæ, 395
— — — — —, diagnosis from exudative tubercular peritonitis, 302
Assyrian foot, 761, 762
Astragalus, dislocation of, 762
—, fracture of, 758
—, injuries of, 761
Auditory nerve injuries through fractured base, 6
— — — — —, focal symptoms in, 47, 48
Axial rotation of intestine, 388, 389
Axilla, abscess of, due to acne pustules or furuncles, 224
—, enlargement of glands, 225
— — —, in cancer of breast, 244, 245
—, hydro-adenitis of, 224
—, pendulous lipoma of, 608
—, phlegmon of, 224

B

Bacilluria, 461
— in cystitis, 495
Back, flat, 559

Back, mobility of, after vertebral injuries, 521
—, muscles of, fibro-lipoma of, 231
— —, fibroma, lipoma, and sarcoma of, 230
—, round, 559
—, skin of, tumours of, and their differentiation, 229-232
Balanitis, 506
Balanoposthitis, 506
Banti's disease, 333, 352
Barlow's disease, signs of diagnostic importance, 109, 544, 696
Base of skull, fractured, symptoms of, 3-7
Bayonet fracture, supra-condylar, in elbow-joint, 592
Bennet's fracture, 621
Biceps muscle, hernia of, 610
Biliary passages, catarrh of, 332
— —, injures of, 263, 264
— —, obstruction of, 340-343
— —, surgical diseases of, 332
— — — —, with peritoneal symptoms, 282, 290
Bites of mucous membrane of cheeks, ulcers due to, 108
Bladder, calculi, 491
— —, aseptic, 491
— —, cystoscopy in, 492
— —, diagnosis from tumours of, 492
— — —, from stone in kidney, 492
— —, in diverticula, 493
— —, infected, 492
— —, in skiagram, 493
— —, obstructing urethra, 458
— —, primary and secondary, 492
— —, symptoms of, 492
—, catarrh of, 494, 495
—, colic of, 464
— disturbaces in spinal injuries, 523
—, ectopia of (vesical), 402
—, examination of, 467, 468
— —, with cystoscope, 469
—, fibroma of, 497
—, fistulæ of, 454
—, foreign bodies in, 497
—, gunshot wound of, 268
—, hæmorrhage from, 463, 498
—, hernia of, 414
—, inflammation of, 494
—, injuries of, 266, 494
—, papilloma of, 497
—, rupture, extra-peritoneal, 266
— —, intra-peritoneal, 267
— —, difference between extra-peritoneal and intra-peritoneal, 267
—, sarcoma of, 497
Bladder, tenesmus of, 456, 488, 496
—, tuberculosis of, 490, 495
—, tumours in muscle of, 497
— —, mucous membrane of, 496
Blindness, unilateral and bilateral, focal diagnosis, 41, 42
Blood, condition of, in Graves's disease, 153
— cysts, in long bones, 700
—, effusion of, diagnostic importance in fractured ribs, 201
— — — — — skull, 3, 4
—, freezing point in kidney disease, 469
— in joints, 722, 724
— tumours at side of neck, 164
—, vomiting of, hysterical, 312
— —, in cancer of stomach, 328
— vessels, injuries of, in warfare, 793
Bone, abscess of, chronic, at lower end of tibia, 750
— —, with sequestrum, in humerus, 575
—, aneurism of, in femur, 701
—, cysts of, in leg, 745, 751
— injuries in warfare, 791
—, perineal fistulæ, in connection with, 451
—, tumours of, in ileo-sacral region, 517
— —, in thorax, 232
Botriomycosis of hand, 639
Brain, abscess of, 18, 19, 20
— —, diagnosis, 18, 19
— —, etiology, 20, 21
— — in temporal lobe after aural suppuration, 33
— —, symptoms, 19, 20
—, angioma of, 57
—, base of, nerves of, 49
—, concussion of, 8, 9, 10
— —, course, 9
— —, diagnosis, 8, 9
— —, symptoms, 9, 10
— — —, local, 15
—, contusion of, 10, 11, 12
— —, diagnosis, 11, 12
— —, symptoms, 11, 12
—, cortical areas of, 46, 47
—, cysts of, 21, 22
—, focal diagnosis, 41
— — —, in tumours, 24, 25
—, gumma of, 19, 23
—, hernia of, 55, 56
— —, differential diagnosis, 57
— —, position of, 55, 56
— —, symptoms of, 56
—, injuries of, 8-18

Brain, tubercle of, 19, 23
—, tumours of, 18
— —, diagnosis, 19
— —, localization, 24, 25
— —, new growths and granulation tumours, 23
— —, diagnosis from abscess of brain, 18, 19
— pressure, 8, 9, 13
— —, general and local, 13
— —, commencing and complete, 14
— — —, diagnosis, 15
— —, symptoms, classical, 14, 15
— — —, local, 14
Branchial cleft cancer, 181
— — fistulæ, 146, 147
— — cysts, 164, 175
— — —, diagnosis of, 175
Breast, *see also* Mamma
—, bleeding, 249
—, cancer of, diagnosis, 244
— —, adhesions to pectoral muscles in, 245
— —, contracting, 242, 243
— —, diminution of the areola in, 242, 244
— —, elevation of nipple in, 242
— —, enlargement of axillary glands in, 244
— —, medullary, 245, 247
— —, prognosis, 245
— —, retraction of nipples in, 244
— —, scirrhus, 239, 241, 249
— —, secondary growths, 231
— —, ulcerating, 245, 246
Bronchial glands, enlargement of, 214, 215
Bronchiectasis, diagnosis, 211
— —, from abscess of lung and tubercular cavity, 209, 210
— diffuse, of right upper lobe of lung, 219
Bronchocele, 150
Bronchoscopy, diagnostic importance of, 118, 124
Brown-Sequard's lesion, 525, 541
Bryant's triangle, determination of, in injuries of hip, 672, 673
Bulbar palsy, dysphagia in, 130
Buphthalmos, diagnosis from exophthalmos, 53
Burrowing abscess, due to spinal caries, 402, 407, 417, 549-552
— —, gluteal, 552
— —, inguinal, 402, 407, 411, 552
— —, perineal, 552
— — in iliac fossa, 551
— — — loin, 550
Burrowing abscess in neck, diagnosis from aneurism, 173
— — — true pelvis, 510
Bursa, iliacus, effusion into, 692
—, mucous, of hand, inflammation of, 643
— — foot, inflammation of, 774
— — knee, inflammation of, 719, 729, 730
— — shoulder, inflammation of, 584
— pre-patellar, tuberculosis of, 729
Bursitis, infra-patellar, 729
— over olecranon, 572, 607
— — tendo Achillis, 774
—, pre-patellar, 729, 730
— —, phlegmonous, 719
—, pre-tibial, 729
—, sub-calcaneal, 774
—, traumatic and tubercular of shoulder-joint, 584, 585
— under callosities of palm, 643

C

CORDE colique, 369
Cæcal region, herniæ in, as causes of intestinal obstruction, 374, 426
Cæcum, movable, 365, 366
Calcanalgia, 775
Calcaneus, compression fractures, 765, 766
—, crush fractures, 765
—, fractures, 763, 764, 765, 766, 767
—, laceration fractures, 763, 764
—, sarcoma of, 773, 789
—, tubercle of, 728, 773
Cancroid of back of hand, 639
— concha of ear, 79
— lips, 72
— nose, 74
Caput obstipum, 196
— —, congenital, 196
Carbuncle of back of neck, 142
Carcinoma, branchiogenous, 181
— of bladder, 497
— breast, 243-247
— —, contracting, 229, 244
— — —, ulcerating, 245, 246
— cervical glands, secondary, 168
— cheek, 69
— clavicle, 226
— concha of ear, 79
— floor of mouth, 108
— — —, perforation of, 148
— hand, 639
— of intestine, 373,
— kidneys, causing intestinal obstruction, 376

Carcinoma of larynx, 126
— —, histology, 128
— —, perforation, 148
— —, position, 128
— lips, 70, 71
— liver, 346
— —, secondary, 346
— lung, 221
— nose, 73
— œsophagus, 137, 138
— ovary, 376
— pancreas, 350
— parotid gland, 179
— penis, 507
— pharyngeal wall, 111
— — —, perforation of, 148
— prostate, 500
— puncta of eye, 74
— pylorus, 328
— rectum, 292, 373, 432, 435
— scalp, seborrhœic, 62
— scrotum, 441
— sigmoid flexure, 375
— soft palate, 111
— stomach, 325
— —, cardiac end of, 326
— —, body of, 327
— submaxillary gland, 177
— testicle, 449
— thyroid gland, 162
— — —, secondary deposits, 163, 164
— tongue, 116, 117
— tonsillar region, 111, 170
— umbilicus, 397
— upper jaw, 90-93
— uterus, 376
Caries of spine. *See* Spinal caries
Carotid gland, new growth of, 182
Carpo-metacarpo-phalangeal joint, dislocation, 618, 619
Cartilage, detachment of, in knee-joint, 711
Cartilaginous tumours in pelvic joints, 512
Catheterization of urinary passages, 466, 467
— — — in ruptured bladder, symptoms, 266
— — —, injuries through, 489
Cauda equina, compression of, irritative symptoms, 530
Cavernitis, chronic, of penis, 505
Cellular tissue, subcutaneous, of finger, inflammation of, 641
Cephalhæmatoma, 3
Cephalo-hydrocele, traumatic, 23
Cerebellar abscess after chronic otitis, diagnosis of, 34
Cerebellar tumours, 24, 25
Cerebello-pontine angle, tumours of, 25
Cerebral injuries, 1, 8
— —, pressure symptoms, 12, 13, 14
— —, symptoms, 8, 9
— localization, 41
— —, auditory nerve disturbances, 47, 48
— —, disturbances of peripheral ocular muscles, 41, 42
— —, facial nerve lesions, 43-47
— —, methods of marking most important motor centres on surface of skull, 46, 50-52
—, new growths in brain, 23, 24
—, paralysis of limbs,
—, speech disturbances, 49, 50
—, visual disturbances, 41, 42
— pressure, 9
— puncture, diagnostic value in brain tumours, 27
Cerebro-spinal fluid, discharge of, from ear and nose in fractures of base of skull, 4
Cervical cord, injuries of, diagnosis of segment, 526
— fistulæ, 145
— —, arising from a goitre, 147
— — —, cancer, 148
— —, branchial-cleft sinuses, 146
— —, complete and incomplete, 146, 147
— —, congenital, 146
— —, median, 146
— —, mouth, larynx, and pharynx, 148
— —, origin, course, and external appearance, 145
— —, position, 147
— —, secretion, 146
— ribs, 149, 173, 182
— —, symptoms of, 182, 183
— spine, injuries of, disturbances of motion in, 526
— — — —, sensation, 528
— — —, irritative symptoms, 529
— veins, dilatation in mediastinal tumours, 213
— vertebræ, caries of, 193, 544-553
— —, contusions, 188
— —, dislocation fracture (complete dislocation), 187, 188, 189, 195
— —, unilateral (rotation-dislocation), 194
— —, fracture of axis, 191
— —, injuries, 187
— —, normal position of atlas and axis, 191

Cervical vertebræ, osteo-myelitis of, 192
— —, sprains, 188
— —, tubercle of, 143, 145, 547, 549
— —, tumours of, 196
Cervico-dorsal scoliosis, with elevation of shoulder, 558
Chancre, hard and soft, 507
—, on the fingers, 644
—, primary, on face, 79
— —, fingers, 644
— —, gums, 109
— —, lip, 69
— —, tongue, 115
— —, tonsil, 110
Cheeks, bites of, 108
—, gangrene of, 99
—, lupus of, 77
—, molluscum contagiosum of, 77
—, rodent ulcer of, 73, 74, 77
—, tumours of, 67
— —, mucous membrane of, 100
Cheyne-Stokes's respiration, in brain-pressure, 13 14
Chimney sweep's cancer of scrotum, 441
Cholangitis, acute, 345
Cholecystitis, acute, 335
— —, differential diagnosis, 335, 336
—, gangrenous, 338
Cholesteatoma of temporal bone, diagnosis, 28, 34
Chondritis of ribs, 227
Chondroma of arm, 611
— cervical rib, 184
— hand, 638
— lower jaw, 95
— mamma, 250
— parotid gland, 179
— spine, 540
— thigh, 700
— tibia, 745
— toes, 787
Chopart's joint, dislocation of, 768
— —, inflammation of, 771
Cicatricial stenosis of bowel, 374, 385
— — pharynx, 130
— — stomach, 310, 324
Circumflex nerve paralysis through dislocation of shoulder, 630
Cirrhosis of liver, hypertrophic, 333
— stomach, 328
Clavicle, carcinoma of, 226
—, dislocation of, 569, 570
—, fracture of, 568, 569
—, osteo-myelitis of, 224
—, sarcoma of, 226
Club foot, 782-786
Club foot, bilateral, with flaccid paralysis, 782, 784
— — —, spastic, 782
— hand, 632, 633
Cochlear nerve, focal symptoms in injury of, 47
Colic, intestinal, 364
— —, in obstruction, 368
Colitis, 360, 368
—, chronic ulcerative, 362
—, muco-membranous (symptoms and diagnosis), 363, 364
Colloid goitre, diffuse, 151, 155
— —, unilateral, 156
Colon, *see* Large intestine
Comminution fracture of head of humerus, 578
— — lower end of ulna, 616
— — os calcis, 765
— — patella, 713, 715
Common bile-duct, closure of, 340
— — — by stones, 340, 341
— — — tumour, 342, 343
— — metastases in, 342
Compression fractures, cause of curvature, 537
— —, diagnosis from lumbago, 520
— — of os calcis, 763, 766
— — scaphoid, 768
— — spinal column, 534-537
— brain, 9
Concretions in stomach, 320
— umbilicus, 402
— urine, 464
Concussion of brain, 8, 9, 10
Condyles of femur, fractures of, 709, 710, 711
— humerus, fractures of, 596, 598
Condylomata, tip of penis, 508
Conglomerate goitre, 156
Conjunctiva, chancre of, 80
Constipation, causes of, 363, 364
—, ascending type, 364
—, painful, 364, 432
Contracture, Dupuytren's, 634, 635
— —, of foot, 781
— in ulnar paralysis, 629, 634
—, posture of spinal column, 564
Contrecoup contusion of brain, 16
— fractures of skull, 6, 7, 16
Contusion, axial, of spinal column, 538
—, fracture of semilunar bone, 620, 621
—, intramuscular osteoma after, 610
— of ankle, 752
— brain, 8, 9, 10
— —, diagnosis, 10, 11

Contusion, cervical vertebræ, 188, 189
— gastro-intestinal canal, 261
— — — —, symptoms, 261
— hip-joint, 661
— knee-joint, 709
— scrotum, 439
— shoulder-joint, 570
— thorax, 201
— urethra, without external wound, 502
Coronoid process of ulna, fracture of, 590, 601
Coxa vara, 691, 679-683
— —, diagnosis between spontaneous and traumatic forms, 667, 682
— —, diagnosis from hip disease, 683
— —, from congenital dislocation of hip, 671, 681
— —, due to rickets, 680, 681
— —, due to injury, 661, 667
— —, false, 680
— —, important diagnostic signs, 681
— — of adolescence, 680, 683
— —, unilateral, limping in, 641
Cranial nerves, injury of, 4, 5, 6
— — —, focal symptoms, 41-52
Craniometry, after Kocher and Krönlein, 50, 51, 52
— — — —, diagrams of, 51, 52
Cretinism, fractures in, 714, 716
Crutch-palsy, 631
Cubitus valgus, 595
Curvature of spine, 555-567
Cystadenoma phyllodes of the breast, 238
Cystic goitre, 155, 156
Cystitis, 461, 494
—, causes of, 494
—, composition of urine in, 462, 495
—, primary, diagnosis of, 494
—, tubercular, 495
Cystoma of jaw, multilocular, 95, 96
— testicle, 448
Cysto-sarcoma phyllodes of the breast, 238
Cystoscopy, 455, 467, 469, 492, 497
Cysts between testicle and epididymis, 446
—, blood, 172
— in true pelvis, 431
—, lymph, 171
— of abdominal cavity, diagnosis from sacculated tubercular effusions, 302, 307
— bones of leg, 745, 751
— brain, traumatic, 21, 22
— branchial clefts, 164, 175
— breast, 240, 241, 242
Cysts of hand, traumatic, 638
— jaw, 92, 94
— kidneys, congenital, 487
— liver, 346, 347
— —, parasitic, 347
— mouth cavity, 100, 101
— neck, congenital, 175
— omental, 307
— pancreas, 350, 351
— prostate and retro-prostatic tissue, 512
— spleen, 353
— thyreo-glossal duct, 164
— thyroid, 155
— umbilicus, 395
— urachus, 397
— vitelline duct, 397

D

DACROCYSTITIS, phlegmonous, 65
Dactylitis, syphilitic, 648
Défense musculaire in abdominal injuries, 262, 274
Degeneration, fibro-epithelial, of mamma, 238
Dental cysts, 91, 92
— periostitis, 64
— sinus, inflammation of jaw in, 86
Derangement, internal, of knee-joint, 709, 711
Dercum's disease, 185
Dermatitis of hand, 640
— —, through drugs, 640
Dermoid fistulæ around coccyx, 450, 517
— of back of neck, 184
— face, 67
— floor of mouth, 97, 140
— lung, 221
— mediastinum, 221
— neck, 175
— pelvic cellular tissue, 512
— penis, 505
— sacral region, 517
— scrotum, 440
— skull, 55, 57
— testicle, 448
— umbilicus, 395
—, sublingual, 101
—, suppurating, 97, 140
—, supra-orbital, 68
Detached fracture of coronoid process of ulna, 590, 601,
— — head of fibula, 717, 718
— — internal condyle of femur, 711

Detached fracture of internal epicondyle of humerus, 590, 593
— — os calcis, 763-767
— — rim of acetabulum, 669
— — spine of tibia, 712, 715
— — styloid process of ulna, 613, 617
— — trochanter of femur, 662
— — tuberosity of humerus, 575, 576, 577
Dextro-position of stomach, 252
Diaphragmatic hernia, after injuries to lung, 203
— —, diagnosis of, 390
Diaphyseal tuberculosis of humerus, 612
— — tibia, 748, 749
— tumours of femur, 705, 706, 707
Diphtheria, bacteriology of, 119
— of larynx, 119, 120
— throat, 119
—, paralysis of palate in, 130
—, secondary symptoms, 120
Diplegia, from spinal cord hæmorrhage, 526
Diploe, sarcoma of, 61
Dislocation, acromio-clavicular, 573, 574
—, axillary, 578
—, central, of femur, 659
—, compression-fracture of spine, 537, 538
— — of cervical vertebræ, 188
—, congenital, of hip, 671-679
— fracture, complete, 535
— — in ankle, 761
— — of cervical vertebræ, 188, 189
— — spinal column, 535
— —, typical, inter-carpal, 620
—, iliac, 655, 656
—, ileo-pectineal of pubic, 657, 658
—, infra-cotyloid, 660
—, obturator, 659
— of ankle, 759-763
— astragalus, 762
— carpo-metacarpo-phalangeal joint, 621
— Chopart's and Lisfranc's joint, 768
— clavicle, 567, 570
— elbow, 595
— —, backwards, 588, 589, 595
— —, forwards, 595, 597
— —, lateral, 596
— —, outwards, 596
— femur, 655
— —, central, 659
— hip, 650, 655-670
— —, backwards, 655, 656
Dislocation of hip, complicated, 656, 657
— —, congenital, 671-679
— —, forwards, 659
— —, inflammatory, 685
— humerus, 540, 573, 574, 578
— inter-carpal phalangeal joints, 618, 619
— knee-joint, 718
— lower jaw, 81
— lumbar vertebræ, 530
— neck, unilateral, 194, 195
— —, complete, 187, 188, 195
— patella, 718
— peroneal tendons, 759
— shoulder-joint, 574, 578
— tibia, 718
— ulna, 589, 595
— vertebral column, 533, 534, 535
— — —, complete, 538
— wrist, 619, 620, 621
—, perineal, 659
—, radius, 597, 598
—, sciatic, 655, 656
—, semilunar bone, 619, 623
—, sub-astragaloid, 762
—, sub-coracoid, 573, 578
—, supra-cotyloid, 660
Diverticula in lower portion of large intestine, inflammation, 292
—, Meckel's, invagination of intestine, caused by, 387
— of bladder, stones therein, 493
— œsophagus, 135, 144, 149, 172
—, perforation of, Meckel's, 283
Dorsal scoliosis, 559-567
— spinal cord, local diagnosis, 526-530
— vertebræ, compression fracture, 534-5
— —, primary scoliosis of, 565
— —, segmental diagnosis of, 527
— —, suppuration, 549
— — —, with burrowing abscess, 549
Duodenal ulcer, diagnosis from cholecystitis, 335
— — — gastric ulcer, 323
— — — intestinal obstruction, 381
— —, perforated, 329
— —, peritonitis in, 282
Dupuytren's contraction, 634, 635
Dura mater, hæmatoma of (local pressure symptoms), 12-15
— —, pedunculated fibroma of, 186
Dysentery, diagnosis from intestinal obstruction, 371
— —, ulcerative colitis in its later stages, 363
Dyspnœa in cerebral pressure, 14

Dyspnœa in diphtheria, 120, 121
— mediastinal tumour, 213
— perforated gastric ulcer, 329
Dysuria, 457

E

EAR, cancroid of, 78, 79
—, concha of, prominence of, in mastoid otitis, 30
—, hæmorrhage from, diagnostic significance in fractured base, 3
—, lupus of, 78, 79
Echinococcus of kidney, 487
— liver, 347
— —, multilocular, 346, 347
— lung, 207, 221
— mediastinum, 221
— spleen, 353
— vertebral column, 542, 543
Elbow glands, inflammation of, 603
— joint, dislocation, 589, 595
— — —, backwards, 595-597
— — —, complete and incomplete, 589, 595, 596
— — —, forwards, 595
— — —, lateral, 589, 596
— — —, outwards, 589
— — fractures at lower end of humerus, 589, 590, 592-600
— — —, coronoid process, 600
— — — head of radius, 599, 601
— — — olecranon, 590, 591
— — — ulna, 590, 600
— —, inflammation, 603-607
— —, injuries of, 587
— — —, examination for, 588
— — — — by inspection, 588
— — — — palpation, 594
— — — — testing movements, 588
— — — — X-ray examination, 600
— —, normal, 591
— —, position of three lines of fracture of, 590
— —, sprain of, 589
— —, syphilis of, 604
— —, tuberculosis of, 604, 605, 606
Elephantiasis of penis, 505
— scrotum, 440
Embolism of mesenteric artery, 281
— lung, 210
Embryoma of testicle, 449
— —, diagnosis from embryoid tumour, 449
Eminentia capitata humeri, abrasion fracture, 599, 600
Emissary mastoid vein, thrombophlebitis of, 35
Emphysema in injuries of thorax, 201
Empyema necessitatis, 222
— of antrum of jaw, 91
— gall-bladder, 343
— lung, 208, 209
— —, scoliosis after, 557
Encephalocele, 56
Encephalomeningocele, 56
Enchondroma, *see* Chondroma
Endothelioma, relation to cutaneous cancer, 79
Enteroptosis, 251-260
—, confirmation by palpation, 254
— — by skiagram, 256
Enterospasm, 279
Epicondyle of humerus, internal, fracture of, 593, 596, 597
Epicondylitis of humerus, 607
Epididymitis, gonorrhœal, 443, **444**, 446
—, syphilitic, 446
—, traumatic, 443
— —, sinuses in, 446
—, tubercular, 446
Epigastrium, acute inflammation, 282
—, chronic abscess of, 392, 393
—, hernia of, 393, 394
—, subcutaneous lipoma of, 393
Epilepsy, diagnosis from hysteria, 37
—, diagnostic significance of course of attacks, 39
— — —, the history, 38
— — —, physical condition in intervals, 39, 40
—, epileptiform attacks in brain tumours, 19
—, Jacksonian, attacks of, in cerebral pressure, 18
— — — cerebral tumours, 19, 40
— —, typical form of, 39
—, surgical significance of the various forms, 37
Epinephritis, 470
Epiphyseal swellings of femur, 701
Epiphysis of tibia, tuberculosis of, 750
—, separation of, at head of femur, 667, 685
— — in the new-born, and in infants with hereditary syphilis, 577
— — radius, 599
Epispadias, 505
Epithelial cysts of neck, congenital, 175
— — —, traumatic, of hand, 638
Epithelioma of face, calcified, 68
Epulis, 102
Erb's paralysis, posture of hand and fingers in, 630

Erysipelas after gunshot wounds, 804
— of face, 63
— scalp, 55
Erysipeloid of finger, 641
Esmarch's paralysis in the upper extremity, 631
Eye muscles, nerves of, injuries to, 6
— —, peripheral derangements of, focal diagnosis, 42-44
— — — —, in Graves's disease, 151
Eyelids, cancer of, at puncta, 74
—, ulcers on, 77
—, xanthelasma of, 68
Eyes, conjugate deviation of, in cerebral pressure, 14, 43
— — — — — —, diagnosis from peripheral derangements of ocular muscles, 42, 43
Exophthalmos, diagnostic significance of unilateral and bilateral, 53, 54
— in Graves's disease, 53, 151
— pulsating exophthalmos, 53
—, surgical, 53
— —, mode of origin, 53, 54
Exostosis of femur, cartilaginous, 700, 701, 702
—, subungual, of foot, 787
Expansile pulsation in aneurism, 172
Extension apparatus of knee-joint, laceration of, 712
— fracture of lower end of humerus, 589, 590, 592, 593, 594
Extra-uterine pregnancy, peritoneal symptoms, 285
Extremities, measurement of the lower, after injuries, 652, 653, 672, 673
—, mobility of, after cerebral injuries, 49
— — — vertebral injuries, 521, 522
—, rigidity of the lower, causing limp, 650
—, sciatica and other painful diseases of the lower, 731-735

F

FACE, angioma of, 68, 69
—, cirsoid aneurism, 68
—, cutaneous warts of, 68
—, dermoid of, 67
— —, supra-orbital, 67
—, epithelioma of, 68
—, erysipelas of, 63
—, inflammatory processes of, 63
—, lipoma of, 67
—, lupus of, 71, 72, 77
—, primary chancre of, 79
Face, sebaceous cyst of, 67, 68
—, syphilide of, 79
—, tumours of, 67
—, ulcerative processes of, 69-80
—, xanthelasma of, 68
Facial nerve injuries, focal diagnosis of, 43-46
— — —, diagram of, 45
— paralysis in fracture of skull, 4, 43, 44
— —, tetanus, 82, 83
False croup, symptoms of, 120
— tail in sacral region, 517
Fat necrosis in pancreatitis, 281
Fatty neck, Madelung's, 185
— tissue, retrobulbar, venous thrombosis of, 65
Femoral hernia, 416-420
— —, abnormalities in, 420
— —, diagnosis of, 419, 420
— — —, from burrowing abscess, 416
— — — distension of saphenous vein, 416
— — — glandular enlargement, 417
— — — pedunculated subserous lipoma, 417
— —, external, 420
— — in women, 418
— — —, with divided sac, 419
— —, pro-peritoneal, 420
— —, strangulated, 419, 428
Femur, Barlow's disease, 696
—, chondroma of, 700
— dislocation, 655
— —, anterior, 659
— —, central, 659, 670
— —, complicated, 656, 657
— —, congenital, 671-679
— —, posterior, 655, 656
—, exostosis, 700, 701, 702
—, fracture of cartilage of, 709, 710
— —, diaphysis, 668
— — — at upper end, 662-666
— — — at lower end, 713, 716, 717
— — —, spontaneous, 706, 707
—, osteomyelitis of, 684, 685, 697, 698, 703, 704
—, sarcoma of, 698, 699, 700, 702
—, tuberculosis of, 686-691
Fibro-adenoma of mamma, 241, 242, 243
— —, phyllodes, 238
Fibro-lipoma of muscles of back, 231
Fibroma of abdominal wall, 398, 399
— arm, 611
— back of neck, 185
— bladder, 497
— hand, 638

Fibroma of knee-joint, 729
— larynx, 128
— leg, 745
— lower jaw, 95
— mouth cavity, 100, 102
— nail-bed of toes, 787
— naso-pharynx, 93, 105, 131
— neck, 176, 180
— pelvis, 511
— sacral region, 517
— scalp, 59
— scrotum, 440
— thigh, 698, 699
— thorax, 230, 231, 232
— tongue, 114
— umbilicus, 395
Fibro-myoma of ligamentum teres, 398
— of the uterus, 306
Fibro-sarcoma of neck, 180
— skin of back, 232
— small intestine, 369
— tibia, 745
Fibula, aneurism of, 745, 751
—, cracking of, with abduction fracture, 755
—, congenital defect of, 779, 781
—, fracture of, 753-757
—, head of, detachment, 717
Finger, anomalies of posture in nerve lesions, 629, 631, 634
—, bent little, 635
—, derangement of movement in tendon-sheath inflammation, 623
—, dermatitis of, 840
—, destruction of, by leprosy, Raynaud's disease and syringomyelia, 645, 646
—, Dupuytren's contraction, 634, 635
—, erysipeloid, 641
—, inflammation of, 643
— —, chronic, 644
— —, subcutaneous cellular tissue of, 641
—, injuries of, 621, 622
— joint, inflammation of, 642
— — —, gonorrhœal, 642
— —, purulent inflammation of bone, 643
—, syphilis of, 641, 644, 645
—, teno-synovitis of, 641, 642, 646
— — —, contracting, 647
— — —, crepitating, 642
— — —, gonorrhœal, 647
—, trigger, 635
—, tuberculosis of, 644, 647, 648
—, tumours of, 636
— —, innocent, 636
— —, malignant, 639
Fissures of fibula, 755
— pelvic bones, 670
— rectum, 434
— skull, 6, 7
— —, course of, 6, 7
— upper jaw, 80, 81
Fistula in ano, 436, 450-453
— —, acquired, inflammatory, 452
— —, congenital, 451
— in connection with goitre, 147
—, urinary, 454
Flat-foot, 776, 777, 778
—, contracture in, 780
—, paralytic, 781
Flexion fracture of lower end of humerus, 596
Flexor tendons of fingers, teno-synovitis of, 646
Focal diagnosis in cerebral diseases, 41
Foot, angioma of, 788
—, bursa of, inflammation, 774
—, carcinoma of, 789, 791
—, chondroma of, 787
—, deformities of, 776-787
—, dislocation of, 759-763
— —, at Chopart's and Lisfranc's joint, 768
— —, Volkmann's, 781
—, epithelioma of, 791
—, fibroma of, 788
—, gout of great toe, 775
—, gumma of, 772
—, habit contracture of, 780, 783
—, inflammation of, 771
— — metatarsus and toes, 775
— — tarsus, 771
—, injuries of, 768-771
—, lipoma of, 788
—, normal, 778
—, perforating ulcer of, 789
—, peri-articular abscess of, 772
—, sarcoma of, 773, 789, 790
—, syphilis of, 725, 791
—, tuberculosis of, 773, 774, 791
—, tumours of, 787
—, ulcers of, 789, 790
Foreign bodies in bladder, 494
— — knee-joint, 710, 711
— — larynx, 123, 124, 125
— — —, dyspnœa in, 124
— — œsophagus, 131, 132, 133
— — pharynx, 131, 132
— — rectum, 437, 438
— — stomach, 320
— — trachea and lungs, 123, 124, 125, 209
— — urethra, 458

Foreign bodies in wounds of warfare, 796
Fork-like posture of hand in fracture of radius, 612, 613
Foveola coccygea, 451
Fracture at ankle-joint, 752, 754-758
— knee-joint, 710-718
—, Bennet's, 621
—, bi-malleolar, 759, 760, 761
—, diacondylar at knee-joint, 675
—, greenstick, 568, 613
— of anatomical neck of humerus, 576, 580
— astragalus, 758
— axis, 191
— capitulum radii, 599, 602
— clavicle, 568-571
— condyles of femur, 715, 717
—, coronoid process of ulna, 590, 600, 601, 602
— epi-condyles of humerus, 593, 596, 597, 598, 603
— femur, at lower end, 713, 715, 716, 717
— —, at upper end, 662-666
— —, shaft of, 668
— —, spontaneous, 706, 707
— fibula, 718, 753, 754, 755
— humerus at lower end, 594-602
— — at upper end, 574-580
— — below tuberosities, 576, 579, 580
— —, condyles of, 593, 596, 597, 598, 603
— —, diacondylar, 590, 602
— — through tuberosities, 575, 576, 577, 578, 579
— laryngeal cartilage, 123
— leg, 713, 715, 716, 752
— lower jaw, 80
— malleoli, 753, 754, 755, 756, 757, 758, 759, 760
— metacarpal bones, 621
— metatarsal bones, 769, 770
— neck of femur, 661, 663
— — —, inter-trochanteric, 658, 663, 664, 667, 671
— — —, per-trochanteric, 663, 666, 668, 671
— — —, subcapital, 663, 665, 667, 671
— — —, subtrochanteric, 663, 664, 668, 670
— neck of scapula, 577, 580
— olecranon, 590, 591
— os calcis, 763-767
— patella, 715
— pelvic fossa, 668, 669
— pelvic ring, 668
Fracture of pelvis, 504, 668
— radius, 612-617
— ribs, 199
— rotula, 590
— scaphoid of wrist, 620
— — of foot, 768
— scapula, 577
— semilunar bone, 620, 621
— sesamoid bone of foot, 771
— skull, 1-7
— —, course of, 6
— —, secondary injuries, 4, 5
— sternum, 200
— sustentaculum tali, 766
— tibia, 715, 716, 717
— —, at lower end, 753, 754, 755
— —, infracondylar, 714, 715
— tuberosity of fifth metatarsal, 771
— upper jaw, 80, 81, 82
— vertebral column, 188-191; 520-537
—, Shepherd's, 758
—, supracondylar, at lower end of humerus, 590, 592, 593, 595, 600
— —, knee-joint, 716, 717
—, supramalleolar, 760
Furuncle in axilla, 224
— of back of neck, 142
— lips, 63, 97

G

GALL-BLADDER, abnormalities of position, 336
— —, empyema of, 343
— — —, source of infection for cerebral abscess, 21
— — fistulæ, 402
— —, gangrene of, 338
— —, hydrops of, 343
— — —, diagnosis from floating kidney, 344
— —, rupture of, 263
Gall-stone colic, 333
— — —, anatomical basis of, 334
— — —, diagnosis from epigastric hernia, 334
— — — — from peritonitis, 279
— — — — renal colic, 334
— stones causing intestinal obstruction, 343, 386, 387
— —, diagnosis of, 334, 335, 339
— —, in small intestine, 292
Ganglion of wrist, 636, 637
Gangrene of foot, diagnosis from sciatica, 733
— lung, 208, 209

Gangrene, senile, 733
—, toes, 728
Gas infection of wounds, 805
Gastralgia, 310
Gastro-intestinal canal, contusion of, 261
— — — —, diagnosis, 262
— — —, gunshot wounds of, 268, 269
— — —, rupture and its symptoms, 261, 262
Gastroptosis, 254, 259, 260
Genitalia, diseases of, in women, examination for, 467
— of hermaphrodite with vagina and testicles in hernial sac, 407
Glanders, ulcers of nose in, 111
Glandular abscesses in neck, 139, 140, 141
— — —, chronic, 143
— swelling in cancer of breast, 244
— — inguinal region, 698
— — — —, diagnosis from hernia, 417
— — in leukæmia and pseudo-leukæmia, 165
— — syphilis, 165
— — tonsillar ulceration, 109, 110
— — tuberculosis, 164, 165
— — of neck, 164, 165
— — —, malignant, 168, 169
Glossitis, symptoms of, 98
Glottis, œdema of, 121, 122
Goitre, aberrant, 104
—, abnormalities in position of, 156
—, circumscribed nodular, 155, 156
—, complications of, 159
—, cystic, 155, 156
—, deep, 156
— —, diagnosis from mediastinal tumour, 213
—, diagnosis from aneurism, 173
—, diffuse colloid, 151
—, external appearances of, 150
—, hæmorrhage in, 159, 160
—, heart, thyreotoxic, 153
—, inflamed, 160, 161
— —, œsophageal stenosis in, 134
— in Graves's disease, 151, 152
—, intra-thoracic, 156, 213, 215, 219, 220
—, malignant, 162, 163
— —, diagnostic signs of, 163
—, pendulous, 156
—, plongeant, 156, 157
—, proliferating (Langhans'), 163
—, retro-sternal, 156
—, retro-tracheal, 156
—, retro-visceral, 156
Goitre, secondary growths, 61, 159
—, skiagram of, 158, 159
—, vascular, 151
Gonorrhœa, diagnosis from sciatica, 731
—, examination of urine in, 461
— of ankle, 772
— epididymis, 446
— finger-joints, 642
— hip-joint, 685
— knee-joint, 720
— pelvis of kidney, 480
— penis, 506
— prostate, 501
— rectum, 432, 433, 437
— shoulder-joint, 585
— tendon sheaths of hand, 647
— urinary passages, 461, 462
— wrist, 624
Gout, 771
— of foot, 771
— hand, 643
Granulation of dorsal spinal cord, 540
—, tubercular, of hand, 638
Granulomata, inflammatory, of brain, diagnosis, 23
— — —, localization, 24, 25
Graser's diverticula, 342
Graves's disease, blood condition in, 153, 154
— —, changes of thymus in, 154
— —, commencing, 151
— —, diagnostic signs of, 49, 151-155
— —, forme fruste, 153
— —, pronounced, 151
Groin, bilocular or communicating hydrocele in, 397
—, burrowing abscess, 397, 408
—, glandular enlargement in, 398
—, lymphadenoma, 398
—, sarcoma of, 398
—, sinuses in, 402
—, strangulation of testicles in, 424
—, testicles in, 406
—, tubercular, 398
—, tumours of, 397, 398
Gumma of arm, 609, 610, 611
— brain, 19, 23
— breast, 237
— foot, 772
— knee-joint, 726
— larynx, 127
—, leg, 746
— liver, 346
— mamma, 236
— mouth cavity, 107, 109, 111
— neck, 145, 149
— palate, 103, 107, 109

Gumma of penis, 507
— pharyngeal wall, 109
— — — causing paralysis, 130
— ribs, 226
— shoulder, 586
— skull, 61, 62
— spinal cord, 542
— sternum, 227
— testicle, 449
— thigh, 699
— thoracic wall, 225
— tongue, 114, 115, 116
— tonsillar region, 109
— vertebræ, 555
Gums, acute circumscribed, and widespread swelling of, 97
—, growths of, 102
—, hæmorrhage from, 108
—, lead line on, 109, 279
—, pus from, 108
—, tubercular, 84
—, ulcers of, 108
Gunshot wounds, infection of, 803

H

HABIT contracture of foot, 780, 783
Hæmatocele of testis, 443, 447
—, retro-uterine, 291
Hæmatoma, extradural, 12, 16
—, intradural, 12, 16
— in vicinity of Broca's convolution, 15
— of ear, 69
— spinal cord, 526
—, peri-tubal, 289, 291
—, peri-urethral, 503
Hæmatomyelia, symptoms of paralysis in, 526
Hæmaturia in renal calculi, 482
— — tumours, 485
— urinary tuberculosis, 489
Hæmophilia, effusion in knee-joint in, 722
—, hæmorrhages of gums in, 108
—, renal hæmorrhages in, 464
Hæmorrhoids, 432, 435
Hair tumour in stomach, 320
Hairy margin of head and face, tumours of, 79
Hallux valgus, 784, 785, 787
Hammer-toe, 784, 787
Hand, abnormal postures of, 629
— — — in fracture of radius, 613, 614
— — — nerve paralysis, 629-632
— — —, position of damage, 630
Hand, angioma, 638
—, botriomycosis of, 639
—, carcinoma of, 639
—, chondroma of, 638
—, deformity of Madelung's, 632, 633
—, dermatitis of, 640
—, epithelial cysts of, traumatic, 638
—, fibroma of, 638
—, ganglion of, 636, 637
—, gout of, 643
—, inflammation of, acute, 640, 642
— — bursæ under callosities, 643
— — bone, 647
— —, chronic, 644
— — skin, 644
— — tendon sheaths, 646
—, injuries of, 612
—, leprosy of, 645
—, lipoma of, 638, 647
—, lupus of, 645
—, œdema of dorsum of, 624, 643
—, osteomyelitis of, 643
—, *post-mortem* tubercle of, 645
—, sarcoma of, 639
—, sebaceous cyst of, 638
—, syphilis of, 648
—, tendon sheath, inflammation of, 624, 646
— — — —, gonorrhœal, 647
— — — —, tubercular, 625, 646
—, tubercle of, 637, 638
— — bone, 647
—, tumours of, 636
— —, innocent, 636
— —, malignant, 639
Head, abnormal posture of, 186
— — —, asymmetrical, 193
— — —, symmetrical, 187, 196
—, rigidity of, painful, 186
— — of gradual onset, 193
— — of sudden onset, 187
—, surgical diseases of, 1
— tetanus, 83
— —, with facial paralysis, 83
— tumours acquired, 59
— — —, innocent, 59
— — —, malignant, 60, 61, 62
— —, congenital, 55
Heart, compression of, by effused blood, 206
—, injuries of, 204
— —, avoidance of probing in, 201
— —, cardiac dulness in, 205
— —, diagnosis, 206
— —, pure, 205
— —, subjective sensations in, 205
— —, with injury to pleura, 206

Heart, injuries of, position and nature of, 204
— — — —, reflex symptoms in, 205
Hemianopia, diagnostic significance of, in cortical tumours, 25
Hemianopsia, diagnostic significance in brain pressure, 15
—, focal diagnosis of, 41, 42
Hernia, abdominal, 399
—, crural, 416-420
—, duodeno jejunal, 390
—, epigastric, 392, 393
— —, diagnosis from biliary colic, 334
—, Hesselbach's, 420
— in cæcal region, 390
—, incarcerated, 389, 422, 425
—, inguinal, 397
— —, external, 403-413
— —, internal (direct), 413
— in linea semicircularis Spigelii, 399
—, intermuscular, 404, 407, 409
—, internal, in cæcal region, 390
— in Winslow's foramen, 390
—, irreducible, 422
—, labial, 405, 407, 411
—, Littré's, 425
—, lumbar, 398
—, multilocular, 395
—, obturator, 389
—, omental, 412
— of diaphragm, 203, 390
— umbilical cord, 394
—, pro-peritoneal, 404, 407, 409
—, scrotal, 411
—, strangulated, 422-430
— —, irreducibility of, 422
— —, condition found at operation, 428, 429
— —, after reduction, 389, 430
— — — —, diagnosis from a strangulated inguinal testicle, 424
— — — — — hydrocele, 423
— — — — — peritonitis, 279
— — — — diagnosis of, 425, 426
— —, fistulæ subsequent to, 402
— —, its position in femoral herniæ, 419, 428
— — — — in inguinal herniæ, 427
— — — — in umbilical herniæ, 428
— —, intestinal obstruction with, 389, 426
— —, reduction en masse, 426, 430
— —, stages of, 429
—, subcutaneous, 404, 407, 409
—, tendency to, 403, 405
—, traumatic, 421, 422
Hernia, umbilical, 394, 395
Hernial sac, appendicitis in, 425, 426
— —, contents of, 427
— — — in male hermaphrodite, 407
— —, peritonitis of, 425
— —, sub-divided, 407
— —, tuberculosis of, 426
— — with peri-hernial fat, 412
Herpes genitalis, 506
Hip, adducted (coxa adducta), 680, 681
— — — —, important diagnostic symptoms, 683
— disease, 684
— —, acute infective, 684, 685, 686
— — — —, consequences of, 685
— —, chronic, non-tubercular, 694, 695, 696
— —, diagnosis of, differential, 691, 692
— — — by gait, 687
— — — painfulness, 691
— — — palpation of joint, 690
— — — posture of leg, 689
— — — skiagram, 692, 693
— — — testing movements of joint, 688, 689
— —, secondary changes in, 693
— —, tubercular, 686-694
— —, variety and degree of, 692
— joint, acute inflammation of, 684
Hip-joint, acute inflammation of, results, 685
— —, arthritis deformans of, 679, 694, 695, 696
— —, chronic inflammation of, 686
— —, contusion of, 661
— — —, diagnosis, 652
— —, deformities of, congenital, 671-679
— — —, non-traumatic, 671
— —, dislocation of, 655
— — —, anterior, 659
— — —, complications, 656, 657
— — —, congenital, 671-679, 691
— — —, inflammatory, 689
— — —, irregular, 656
— — —, posterior, 655, 656, 657
— —, examination of, 651
— — — by inspection, 651
— — — palpation, 654
— — — testing mobility, 654
— —, fracture of, 650, 661-670
— —, gonorrhœa of, 685
— —, injuries, table of, 670, 671
— —, osteomyelitis, 684
— —, rheumatism of, 691
— —, sarcoma in vicinity of, 511
— —, senile disease of, 695

Hip-joint, skiagraphy of, 692
— —, sprain of, 661
— —, tuberculosis of, 686
Hirschsprung's disease, 304
— —, diagnosis from abdominal tumour, 304
Hodgkin's disease, *see also* Pseudoleukæmia
— —, signs of, 167, 168, 169
Horns, cutaneous, 79
— —, on heel, 788
Hour-glass stomach, 322
— — —, skiagrams of, 316
Humerus, dislocations of, 572, 573
— —, varieties, 573, 574, 578
—, fractures at lower end of, 590-600
— — upper end of, 574-580
—, muscular hernia over, 610
—, myositis ossificans over, 610
—, osteomyelitis of, 611
—, sprain of, 580
—, tumours of, 607
— —, bone, 611
— —, muscles and nerves, 607, 608
— —, skin and subcutaneous tissue, 607
Hydatids, *see* Echinococcus
Hydro-adenitis of axilla, 224
Hydrocele, bilocular in inguinal canal, 397, 410, 411
—, communicating, 397, 410, 411
—, diagnosis from strangulated hernia, 423
— of spermatic cord, 410, 411, 441
— women, 398
— testis, 410, 411
—, relation of vaginal process of peritoneum, 410
Hydrocephalus in meningocele, 57
Hydro-myelomeningocele, 515
Hydronephrosis, closed, 476
— —, diagnosis from ovarian tumour, 307
—, intermittent, 477, 478, 489
—, open, 476
—, remittent, 477
Hydrops of bursa iliaca, 692
— gall-bladder, 343
— —, diagnosis from movable kidney, 344
— knee-joint, 721
— —, intermittent, 721
— —, traumatic, 723
— —, tubercular, 722, 723
— prepatellar bursa, 708
— subdeltoid, diagnosis from effusion into shoulder-joint, 581, 582
Hygroma of neck, congenital, 171
Hyperextension fracture, 592, 595
Hyperkeratosis of hand, 639
Hypernephroma, 487
—, secondary, 216
Hyperthyroidism in Graves's disease, 152, 153
Hypochondrium, inflammatory foci in, 282
Hypospadias, 505
Hypothyroidism, predisposition to fracture of cartilage of femoral condyles in, 710
Hypertrophy of bronchial glands, diagnosis from mediastinal tumours, 214
— of prostate, 498, 499, 500
— spleen, 352
— thymus, 214
— —, diagnosis, 214
— thyroid, 150
Hysteria, abdominal pain in, 278
—, diagnosis, from epilepsy, 37
— — hip disease, 692
— — intestinal obstruction, 391
— — paralysis, 583

I

Ileo-cæcal region, tumours of, 397
Ileo-sacral tuberculosis, 550
— — —, burrowing abscess, 551, 552
Incised wounds of abdomen, 269
— — thorax, 202
Incontinence of fæces, 524
— urine, 459
—, paradoxical, 459, 524
Infantile paralysis, club-foot following on, 782
— —, spinal, diagnosis from congenital dislocation of hip, 679
— —, unusual localization of, 399
Infarct of bowel, 381
— kidneys, 481
— testicle, 445
Infiltration, diffuse gummatous, of tongue, 116
Ingrowing toe-nail, 791
Inguinal abscesses, 552
— herniæ, 403, 413
— —, bladder in, 414
— —, diagnosis of external, in absence of swelling, 404
— — —, in the presence of a swelling, 406
— — —, labial and scrotal herniæ, 411
— —, differential diagnosis from femoral hernia, 410, 411, 419

Inguinal hernia, differential diagnosis from burrowing abscess, 416
— — — — varix of saphenous vein, 416
— — — — of external and internal, 414
— —, external, 403-413
— — in women, 406
— —, intermuscular, 404, 407, 409
— —, internal, 413-416
— —, properitoneal, 404, 407, 409
— —, relations of, to abdominal wall, 405
— — —, to vaginal process and hydroceles, 410
— —, scrotal, 411, 412
— —, site of strangulated, 427
— —, subcutaneous, 404, 407, 409
Intercarpal joints, dislocation of, 618, 619
Intestinal crises, tabetic, diagnosis from obstruction, 381
— distension, abnormal, with visible and palpable contractions of bowel above, 368, 369
— —, murmurs, at seat of, 369
— murmur of stenosis, 369
— obstruction, 368
— —, acute, 380
— — —, causes, 385
— — — — (axial rotation), 388
— — — — (bands and kinks), 385, 386
— — — — (gall-stones), 386, 387
— — — — (intussusception), 387
— — — —, position of obstruction, 381, 382
— — — — (strangulation of an internal hernia), 389, 390
— — — —, symptoms, 380, 381
— — — —, varieties, 384, 385
— —, arterio-mesenteric, 385
— —, chronic, 368
— — —, appearance of bowel in, 368, 369
— — —, condition of stools in, 370, 371
— — —, through external compression, 375
— — —, position of stenosis in, 372
— — —, variety and cause of stenosis in, 372, 373
— —, combined with external hernia, 426
— —, diagnosis from cholecystitis, 338
— — — perforative peritonitis, 381
— — — uræmia, 383
Intestinal obstruction, diagnosis from peritonitis, 274, 280
— —, effect on general condition, 371
— —, hysterical, 391
— — in appendicitis, 358
— —, intermittent, 376, 384
— — in tubercular peritonitis, 302
— — of sudden onset, 384, 385
— —, spastic, 390
— —, transition of incomplete to complete, 384
— rigidity, 369
— stenosis, cicatricial, 374
— —, after reduction of a strangulated hernia, 374
— —, condition found on palpation, 369
— —, concentric, in cancer, tubercle and syphilis of bowel, 372
— —, condition of stools in, 370, 371
— —, general condition in, 371
— — of gradual development, 368
— — large intestine, 370, 372
— — small intestine, 371
— —, skiagraphy in, 376-379
— — through tumours in bowel, 372, 373
Intestine, abnormalities of position, 252
— —, decussation of large and small intestine in, 253
— —, position of appendix in, 253
— —, principal varieties, 252, 253
—, appearance of, in strangulated herniæ, 429
—, axial rotation of, 388, 389
—, carcinoma of, 372, 373
—, fibroma of, 374
—, fistulæ of, 402
—, hæmorrhage of, 261
—, herniæ of intestinal wall, 426
—, infarction of, 381
— injuries, 261, 262, 263, 374
— —, clinical picture of, 262, 263
—, intussusception of, 292, 387
—, lipoma of, 374
—, myoma of, 374
—, obstruction of, 368-391
— paralysis in injuries to spinal column, 524
—, sarcoma of, 374
—, syphilitic stricture of, 374
—, tuberculosis of, 303
— —, diagnosis from cancer, 372, 373
—, ulcers of, 374, 381
Intussusception of bowel, 387
— —, diagnosis from tumour, 305
Iodoform dermatitis, 640
Isthmus faucium, inflammation of, 98

J

JACKSONIAN epilepsy, 18, 39, 40
Jaundice, catarrhal, 332
— — in cholecystitis, 335
— —, infective, 338
— — through obstruction of common bile duct, 340
Jaw, actinomycosis, 83, 86, 89
—, ankylosing, 84
—, antrum of, empyema of, 91
— —, inflammation, 63
—, arthritis, 84
—, cancer of, 90, 91, 92
—, cysts of, 91, 92
—, dislocation of, 81, 82
—, fractures of, 80, 81
—, inflammation, acute, 83
— —, chronic, 83, 84
—, lower, chondroma of, 95
— —, dislocation of, 82
— —, fibroma of, 95
— —, fractures of, 80
— —, multilocular cyst, 87
— —, osteoma of, 95
— —, sarcoma of, 95, 96
— —, tumours of, diagnostic signs, 93-96
—, multilocular cystoma of, 95
—, osteoma of, 95
—, osteomyelitis, 84, 86, 140
—, periostitis, 63, 64, 83, 86, 91, 92, 140
—, phosphorus necrosis of, 89
—, sarcoma of, 95, 96
—, tuberculosis of, 65, 84, 87, 88, 89
—, upper, carcinoma of, 91
— —, empyema of, 91
— —, fractures of, 80, 81
— —, periostitis of, 63, 91, 92
— —, sarcoma of, 91, 92
— —, tuberculosis of, 92
— —, tumours of, 90
— — —, innocent, 93
— — —, malignant, 91, 92
Joints, injuries of, in warfare, 792

K

KIDNEY, abnormal mobility of, 473
— abscess, 481
— calculi, 482, 483, 484
— —, composition, 483
— — in renal tuberculosis, 490
— —, primary, 482
— —, secondary, 483
— —, X-ray diagnosis, 483
—, carcinomata, 485
Kidney, colic of, 464, 482, 490
— —, diagnosis from biliary colic, 337
— — — peritonitis, 282
—, cysts of, 487
—, displacements of, 254
— —, acquired, 473
— —, congenital, 251
— —, diagnosis from distended gall-bladder, 344
—, examination of functions of, 469
— hæmorrhage, 265, 463
— — in hæmophilia, 464
— — tuberculosis of, 489
— — tumours, 485
—, hydatid of, 487
— infarcts, 481
—, inflammation around, 266, 282, 470
—, injuries of, 265
— —, extra-peritoneal, 265
— —, intra-peritoneal, 265
—, pelvis of, inflammation of, 480
— —, new growths of, 485
— —, suppuration of, 478
—, sarcoma of, 486, 487
—, spontaneous suppuration of, 479
— — —, bacteriology of, 479, 480
— — —, diagnosis, 480
—, tuberculosis of, 472, 478, 488
— tumours mixed, 306, 487
— — of, 485
— —, diagnosis, 485, 486, 487
— —, displaced, 487
Klumpke's paralysis, posture of hand and fingers in, 630, 631
Knee-joint, abscess, peri-articular, 721
— —, aneurism, 730
— —, bursal inflammation, 708, 729, 730
— —, chronic rheumatism of, 721, 722, 723
— —, contracture of, 728
— —, contusion of, 709
— —, detachment of internal semilunar cartilage, 711
— — — — — —, its incarceration, 711, 720
— — — — cartilage of femur in, 710
— —, dislocation of, 718
— —, effusion, acute, 708, 720
— — —, chronic, 721
— — —, idiopathic, 721
— — —, traumatic, 723
— —, fibroma of synovial membrane, 729
— —, foreign body in, 711, 720
— —, fractures of, 709-717
— —, fungating, 726

Knee-joint, gonorrhœa of, 720, 723
— —, gumma of, 726
— — in hæmophilia, 722, 723, 728
— —, injuries of, 708-718
— —, intermittent hydrops of, 720
— — — —, tubercular, 721, 724
— —, lipoma arborescens of, 728
— —, normal, 725
— —, osteomyelitis of, 720, 723
— —, rigidity of, 728, 729
— —, sarcoma of, 726
— —, sesamoid bone, 710, 711
— —, sprain, 709, 711, 712
— — —, results of, 712
— —, syphilis of, 722, 723
— —, tuberculosis of, 723-729
— — —, ankylosing, 728
— —, tumours and allied structures in neighbourhood of, 729, 730

L

Labour, obstruction of, by pelvic tumours, 512
Labyrinth, injuries of, 6
Lachrymal glands, inflammation of, 65
Large intestine, displacements, chief varieties, 253
— — — in skiagram, 257, 258, 259
— —, disturbances of function, with anatomical changes, 361
— — — —, without typical anatomical changes, 363
— —, diverticula of (inflammation), 292
— —, stenosis of, 368-375
— — —, diagnosis from stenosis of small intestine, 382-383
— —, syphilis of, 362
— —, tubercle of, 362
Laryngoscopy in laryngeal diseases, 118
Larynx, carcinoma of, 126, 127
—, circulatory disturbances of, 122
—, diphtheria of, 119
— —, diagnosis from lacunar tonsillitis, 119, 120
— — — pneumonia, 120
— —, secondary symptoms, 120
—, false croup, 120
—, fibroma of, 128
—, foreign bodies in, 123, 124
—, fracture of cartilage of, 123
—, gumma of, 127
—, inflammation of, 119
—, injuries of, 123
Larynx, œdema of, 122, 123
— —, angioneurotic, 122
—, papilloma of, 128
—, stenosis of, 121
—, surgical diseases of, 118
— — —, acute, 119
— — —, chronic, 125
—, syphilis of, 125, 126, 127
—, tubercle of, 126, 127
—, tumours of, 128
—, ulcers of, 122, 127
— —, differential diagnosis, 127, 128
— —, position, 126
Leg, abscess of bone of, 750
—, bony cysts of, 745, 751
—, carcinoma of, 734, 739, 741
—, chondroma of, 745, 750
—, diffuse inflammation, 746
—, dislocations of, 759-763
—, exostosis of, 750
—, fibroma and fibro-neuroma of, 745, 750
—, fractures of, 752-759
—, gumma of, 748
—, localized inflammation, 748
—, osteomyelitis of, 743-748
—, sarcoma of, 746, 748
—, swellings and tumours of, 741-751
—, syphilis of, 746, 748, 749
—, tuberculosis of, 748, 750
—, ulcers of, 735
— —, malignant, 734
— —, syphilitic, 735, 737
— —, varicose, 735, 736
—, varicose veins of, 733, 737
Length measurements of lower limbs after injuries, 653, 654
Leprosy of hand, 645
— of nose, 111
Leucocytosis in abdominal disease, 277
Leukæmia, glandular swelling in, 165
—, hæmorrhage from gums in, 108
—, pharyngeal swelling in, 103
—, splenic enlargement in, 352
Leukoplakia of tongue, 112
Ligamentum patellæ, detachment of, 715
— teres, fibromyoma of, 398
— tibio-fibulare anticum, laceration of, 758
Limping, 650
—, intermittent, 733
—, painful, 651
—, paralytic, 650, 677
— through rigidity of limbs, 651
— — shortening, 650
—, unilateral, 681, 683
—, voluntary, 687

Lipoma in femoral canal, 416
— — —, diagnosis from hernia, 417
— of abdominal wall, 393
— axilla, 608
— back, 229, 230
— —, periglandular, 185
— breast, 250
— epigastrium, 393
— —, subserous, 393
— face, 67
— floor of mouth, 100, 101
— foot, 788
— hand, 638, 647
— knee-joint, 728
— lumbar region, 399
— neck, 169, 176, 185
— pharynx, 106
— sacral region, 517
— shoulder, 608
— spermatic cord, 412, 413, 442
— thigh, 698
— tongue, 114
— upper arm, 607, 608
—, perihernial, 417
Lips, cancer of, 71, 72, 107
—, furuncle of, 63, 97
—, gangrene of, 99
—, primary chancre of, 69
—, tumours of mucous membrane of, 100, 107
Lisfranc's joint, dislocation of, 768
— —, inflammation of, 772
Little's disease, spastic club-foot after, 782
Littré's hernia, intestinal obstruction in, 425
Liver, abscess of, 282, 347
—, acute yellow atrophy, 332, 333
—, adenoma of, 346
—, carcinoma of, 346
—, cirrhosis of, 333
—, constricted lobe of, 325
—, cysts of, 346
— —, parasitic, 347
—, diagnosis of cirrhosis from tubercular peritonitis with effusion, 300
—, displacements of, 252, 254
—, floating, 254, 345
—, gumma of, 346
—, hydatid cyst of, 347
— — —, multilocular, 347
—, injuries of, 263, 264
—, tumours of, 345
— —, primary, 346
— —, secondary malignant, 346
Lordosis of spine, 559
Lumbago, 518
—, course of symptoms, 519
Lumbago, diagnosis from renal tumour, 485
—, rheumatic and traumatic, 518
Lumbar kyphosis, 559
— lordosis, 559
— — in congenital dislocation of hip, 675, 676
— pains in renal disease, 485, 490
— puncture in cerebral pressure, 16
— — — — tumours, 27
— — meningitis, 36
— region, abscess, perinephritic, 398
— —, areas of inflammation in, retroperitoneal, 282
— —, burrowing abscess of, 398
— —, false hernia, 399
— —, hernia of, 399
— —, lipoma of, 399
— —, pigmented nævus of, 232
— —, reflex muscular rigidity in unilateral renal injury, 265
— scoliosis, 559-564
— vertebra, caries of, 544-553
— —, compression fracture of, 533
— —, determination of injured, 532
— —, dislocation of, 533
Lumbo-dorsal scoliosis, 565
Lumbo-sacral cord, segmental diagnosis of lesions, 527
— — —, symptoms of injury of, 530
— — —, types of paralysis, 530
Lung, abscess of, 208, 209
— — in skiagram, 209
—, actinomycosis of, 212, 223
—, bronchiectasis of, 211
—, cancer of, 221
— —, metastatic, 220, 221
—, chondroma of, 221
—, dermoid of, 221
—, embolism of, 210
—, empyema, 208
—, foreign bodies in, 125, 209
—, gangrene of, 208, 209
—, hydatid of, 221
—, injuries of, 201
— —, diagnostic signs of, 202
— —, secondary injuries, 203
—, sarcoma of, 221
—, surgical diseases of, 207
—, tubercular cavities in, 211, 223
—, tumours of, 213, 221
— —, differential diagnosis, 221
Lupus of cheek, 77
— of face, hypertrophic, 72, 78, 79
— hand, 645
— nose, 71, 75
Lymphadenitis, acute submaxillary, 97, 140

Lymphadenitis in neck, 164, 193
— —, malignant, 168
—, phlegmonous submental, 97, 140
Lymphangioma of ear, 68
— floor of mouth, 97, 101
— neck, 170
— sacral region, 517
— scrotum, 440
— thigh, 698
— thorax, cystic, 230
— tongue, 113
Lymphangitis of arm, tubercular, 604
Lymphatic cysts of neck, 170, 171
— gland, enlargement in axilla, 225
— — — cancer of breast, 244
— — — inguinal region, 398, 417
— — — neck, diagnostic significance, 165
— — —, sarcoma in neck, 180, 181
— — — thigh, 698
— — — ulcers of tonsil, 109, 110
— — — causes of, 164, 165
Lymphoma, malignant, 166, 167, 168
— —, diagnosis from lymphosarcoma, 181
— of neck, tubercular, 165
Lymphosarcoma of neck, 181
— lung, 221

M

MACROGLOSSIA, 113
Madura foot, 772
Malarial spleen, 352
— —, rupture of, 263
Malleoli, fractures, 752-761
— —, position and direction of lines of fracture, 753
Mamma, abscess of, 234, 235, 236
— —, retro-mammary, 236
—, actinomycosis of, 236, 237
—, carcinoma of, 243-247
— —, scirrhus, 239, 241, 245
— —, secondary, 246
— —, ulcerating, 245, 246
—, chondroma of, 250
—, cysto-adenoma phyllodes of, 238
—, cysto-sarcoma phyllodes of, 238
—, cysts of, 240, 241, 242
— —, solitary, 241
—, fibro-adenoma of, 241, 242, 243
—, giant growth of, 247
—, gumma of, 236
—, inflammation of, 235
— —, acute, 235
— —, chronic, 236
—, lipoma of, 250
—, sarcoma of, 247
Mamma, sarcoma of, secondary, 248
—, tuberculosis of, 236, 237
—, tumours of, 238
— —, isolated, 239
— — of the male breast, 249
— —, multiple, 239
— —, types of, 238
Manubrium sterni, osteomyelitis of, 141
— —, *see also* Sternum
Manus valga, 632
— vara, 632
Mastitis, acute, 235
—, chronic cystic, 237
— neonatorum, 235
—, puerperal, 235
Mastoiditis, brain symptoms of, 27, 29, 30, 31
Mastoid process, inflammation of, 28
Median nerve paralysis, posture of hand in, 629
Meckel's diverticulum, perforation of, 283
Mediastinum, dermoid of, 207
—, hydatid of, 221
—, injuries of, 202
—, phlegmon of, 134, 141
—, sarcoma of, 221
—, tumours of, 212, 217
— —, malignant, 220, 221
—, tumours of, malignant, diagnosis from deep and intrathoracic goitre, 219, 220
— — — — enlargement of bronchial glands, 214
— — — — — of thymus, 214
— — — — innominate and aortic aneurisms, 214, 215, 217, 218
Medullary cancer of breast, 247
— — penis, 508
— sarcoma of upper end of tibia, 726
Meningitis, diagnosis of, 36
—, purulent, resulting from otitis media, 32, 33
—, serosa circumscripta spinalis, 543
Meningocele, 57, 184, 515, 516, 517
—, spurious, 56
Meningoencephalocele, 184
Menstruation, intra-abdominal, 280
—, painful, diagnosis from peritonitis, 280
Meralgia paræsthetica, 734
Mercurial stomatitis, 84, 99
Mesenteric cysts, 307
— vessels, thrombosis of, 374
— — —, diagnosis from ileus, 381
— — — — peritonitis, 281
Mesentery, common, 252

Mesentery, detachment of, 374
—, ileo-cæcal, 252
Metacarpus, fracture of, 621
—, osteomyelitis and periostitis of, 643
Metatarsus, fracture of, 769, 770, 771
—, inflammatory process of, 775
—, tumours of, 787
Micturition, disturbances of, 456
Middle ear, inflammation, purulent, brain complications of, 27-36
Mikulicz's disease, 176
Military surgery, 791
Mind blindness, 42
Mixed tumours of kidneys, 306, 487
— — palate, 103
— — parotid, 179
— — submaxillary region, 177
Molluscum contagiosum of face, diagnosis from cutaneous cancer, 77, 78
Motion, power of, after cerebral injuries, 46, 47
— — — vertebral injuries, 521, 522
Mouth cavity, acute swelling in floor of, 96, 97, 98
— —, angina of, 98
— —, angioma of, 100
— —, cancer of, 108
— — —, perforating, 148
— —, cysts of, 100, 101
— —, dermoid of, 101, 102
— — —, suppurating, 97
— — —, sublingual, 101
— —, examination of, in dysphagia, 131
— —, fibroma of, 100, 102
— —, gangrenous stomatitis of, 99
— —, gumma of, 103, 107, 109
— —, hæmorrhage from, due to fractured base of skull, 3
— —, inflammatory processes in, 96-99
— —, lipoma of, 100, 101
— —, lymphadenitis of, 97, 98
— —, lymphangioma of, 97, 101
— —, noma, 99
— —, primary chancre of, 109, 110
— —, salivary glands inflammation, 97
— —, tubercle of, 107, 109
— —, ulcers of, 107
Mucous colic, diagnosis from peritonitis, 279
— cysts of mouth cavity, 100
— polypus of nose, 106
— — pharynx, 105
— — rectum, 433
Muscle of thigh, osteoma of, 199
Muscles of neck, gumma in, 149
— thigh, gumma in, 699
— thorax, gumma in, 225
Muscular angioma of arm, 608
— — thigh, 699
— atrophy, progressive, with lordosis, 524, 556, 675, 679
— hernia of thigh, 699
— — upper arm, 609
— paralysis of abdomen, localized, 398
— tuberculosis of abdominal wall, 398
— — arm, 608
— — neck, 149
— — thigh, 699
— — thorax, 225
Myelitis, chronic, 542
—, traumatic, 528
Myelo-cystocele, 515
Myelo-meningocele, 515, 516, 517
Myoma of bladder, 497
— intestine, 374
— uterus, 306, 498
— —, constipation in, 431
Myositis in wry neck, 193, 197
— of sterno-mastoid, 141
— ossificans of arm, 610

N

Nævus of abdominal wall, becoming sarcomatous, 399
—, pigmented, 232
—, vascular, 58
Nail-bed, inflammation of, 641
— —, syphilitic, 642
—, tumours of, 791
—, ulcers of, 791
Naso-pharyngeal fibroma, 93, 105, 131
— — polypi, 104, 105, 131
Neck, abscesses of, 139, 141, 550
— — after aural suppuration, 33
— —, chronic, 143, 144
— — due to spinal caries, 549
— — —, tubercular, 143, 148
—, actinomycosis of, 144, 149
— — with sinus formation, 144
—, aneurism of, 149, 172, 173
—, back of, abscesses in, 142, 143
— —, carbuncle of, 142
— —, cerebral hernia in, 56, 57, 184
— —, dermoids in, 142, 184
— —, fibroma of, 185
— —, lipoma of, 142, 184, 185
— —, rigidity of, with gradual onset, 193
— — —, with sudden onset, 187

Neck, back of, sarcoma of, [illegible]
, sebaceous cysts of, 184
, blood cysts of, 172
, branchiogenetic carcinoma of, 181
, cavernous angioma of, 172
, congenital hygroma of, 171
, cysts of, 170, 171, 174, 175
, dermoid of, 175
, diffuse inflammations of the lymphatic glands, 169
, enlarged glands of, 140, 157, 164
, fibroma of, 170, 180
, fibro-sarcoma of, 180
, gumma of, 145, 140
in muscle of, 140
, inflammatory processes of, 145
, lipoma of, 170
, lymphangioma of, 170, 171
, lymphatic cysts of, 171
glands of, swelling, 110, 164, 165, 167, 168
, malignant, 168
, lymphosarcoma of, 181
, neuro-fibroma of, 180
, phlegmon of, 134, 139, 141
, pseudo-tumours of, 148
, sarcoma of, 180, 181
, deep, 171
, vascular, 171
, sebaceous cyst of, 175
, surgical diseases of, 118
, syphilis of, 145, 165
, tubercle in muscle of, 140
of, 145, 148, 165
, tumours of, 148
back of, 184
side of, 164, 180
, in anterior triangle, 140, 164
, solid, 170
, with liquid contents, 169
Nephritis, chronic haemorrhagic, 404
Nerves of base of skull, 40
Nerve injuries in warfare, 704
Neuralgia of anterior crural nerve, 744
due to cervical rib, 184
external femoral cutaneous, 734
inferior dental nerve, 80, 94
infra-orbital nerve in malignant growths of upper jaw, 90
obturator nerve, 744
sciatic nerve, 741
, peri-articular, of hip, diagnosis from hip disease, 692
Neuro-[illegible] of arm, [illegible]
back, 244
leg, 742, 744, 745
neck, 180
Neuro-fibroma, [illegible]
Neuroma of arm, [illegible]
Nipple, retraction of, [illegible]
Noma of lips and cheeks, [illegible]
Nose, cancer of, 72, 73, 74, 75, [illegible]
, glanders of, 111
, haemorrhage from, in fractured base of skull, diagnostic significance of, 3
, leprosy of, 111
, lupus of, 71, 75
, hypertrophic forms, 72
, mucous polypi of, 106
, rhino-scleroma of, 111
, sarcoma of, 106
, sebaceous cyst at root, 68
, syphilis of, 71, 76
, tubercle of, 111
Nystagmus, local diagnostic significance of, in brain lesions, 42, 43

O

Occipital bone, cerebral hernia at, 56
, osteo-myelitis of, 142
lobe, tumours of, 25
Oculo-motor nerve, injuries of, 5
Odontomata, 95
Oedema of back of hand, 623, 644
glottis, 121, 122
larynx, angioneurotic, 122
Oesophagus, carcinoma, 137, 138
, compression, 135, 136
, difficulties in swallowing in neighbourhood of, 132-139
, diverticula of, 136, 144, 149, 172
, foreign bodies in, 131, 133
, diagnosis of, 132
, removal of, 134
, injuries of, 204
, spindle shaped dilatation of, 136
, stenosis of, 135
, stricture of, syphilitic, 137
, corrosive, 134, 137
Olecranon bursa, inflammation of, 607
, fracture of, 500, 501
, tubercle of, 607
Oliguria, 400
Omental cysts, 307
hernia, 412
, strangulated, 427, 447, 448
tumours, 307
, torsion of, and its symptoms, 381, 427
Onychia maligna, [illegible]
Optic nerve, injuries of, 5
, localisation, [illegible]

Optic neuritis, diagnostic significance in cerebral abscess, 19
— — — — — — tumours, 18, 19
Orbit, angioma of, 53
—, injuries of, 4
—, osteoma of, 53
Orchitis, 443
—, metastatic, 444
Os calcis, *see* Calcaneus
Osteoma of arm, traumatic, 610
— cervical rib, 184
— lower jaw, 95
— orbit, 53
— skull, 59, 61
— thigh, 700, 701, 702
— thorax, 232
— vertebra, 540
Osteomyelitis of arm, 610, 611
— clavicle, 141, 224
— femur, 684, 697, 703, 704
— finger, 642, 643
— hand, 643
— hip-joint, 684, 685
— humerus, 611
— jaw, 86, 140
— knee-joint, 720, 723
— occipital bone, 142
— ribs, 224
— scapula, 224
— shoulder-joint, 587
— skull, 55
— spine, 192, 555
— sternum, 141
— thigh, 684, 685
— —, chronic, 703, 704, 705
— tibia, 746, 747, 748
Osteoporosis in fracture of elbow, 628
— of bones of foot, 773
— carpus, 628
Ostitis fibrosa of femur, 701
— — finger, purulent, 643
— — humerus, 610
— of mastoid, prominence of concha in, 29, 30
— ribs, chronic, 227
Os trigonum, 756, 758
Othæmatoma, 69
Otitis media, chronic, cerebral complications of, 20, 27-36
— — —, symptoms of, 28, 29, 30
Ovarian cysts, 307
— —, diagnosis from hydronephrosis, 307
— —, torsion of, 284, 331, 381
— hernia, diagnosis of, 406
Ovary, cancer of, causing intestinal obstruction, 376
—, fibro-sarcoma of, 306
Ovary, strangulated, 427

P

PACHYMENINGITIS, hæmorrhagic, diagnostic signs of, 18, 21, 22
—, hypertrophic, of cord, 541, 542
Paget's disease, 245
Palate, abscess of, originating in root of tooth, 104
—, acute inflammation of, 123
—, carcinoma of, 107
—, defects in, 130
—, paralysis of, 130
—, polypi of, 104
—, scar development in, 130
—, syphilis of, 107
—, tubercle of, 107
—, tumours of and their diagnosis, 103, 104
Palati, velum, ulcerative processes on, 107
Palm, callosities of, with dilatation of mucous bursæ beneath, 643
—, lipoma of, 638
Palmar aponeurosis, Dupuytren's contraction of, 634, 635
Panaritium (whitlow), 640-643
Pancreas, cancer of head of, 350
—, cysts of, 351
—, diagnosis from cholecystitis, 336
—, hæmorrhage of, 281, 282, 348
— —, intestinal paralysis in, 381
—, stones in, 350
—, surgical diseases of, 348
—, tumours of, 350
Pancreatitis, acute, 348
—, chronic, 349
— —, fat necrosis in, 281
—, diagnosis from cholecystitis, 336
— — ileus, 381
— — perforated gastric ulcer, 331
— — peritonitis, 281
—, urinary reaction in, 349
Papilloma of bladder, 497
— larynx, 128
— palate, 104
Paralysis in cerebral pressure, 13, 15, 16
— congenital dislocation of hip, 678, 679
—, complete injuries of spine, 525, 526
—, ischæmic, 631
—, partial injuries of spinal cord, 525
— of arm, diagnosis from traumatic neurosis, 577
— hand and fingers after nerve lesion, 629-632

Paralysis, palate, 130
—, types of, 530
Parametritis, 289, 290
Paranephritis, 470
Paraphimosis, 505, 506
Paraplegia dolorosa in compression of cauda, 530
—, spastic, occurrence of, 542
— — — in spinal caries, 553
Parathyroids, 182
Paresis of shoulder muscles, diagnosis of, 577
Paronychia syphilitica, 641
Parotid, abscess of, 66, 67
—, cancer of, 179
—, chondroma of, 179
—, mixed tumours of, 179
—, sarcoma of, 179, 180
—, tubercle of, 178, 179
Parotitis, 66, 140
—, epidemic, 66
Patella, dislocation of, 718
—, fracture of, 713, 715
—, laceration of ligamentum patellæ, 715
—, riding of, in articular effusion, 708
—, sarcoma of, 729
Patellar reflexes in vertebral injuries, 524
Pelvic cellular tissue, tumours of, 512
— — —, dermoids of, 512
— cellulitis, 285, 289
— viscera, surgical diseases of, 251, 283-293
Pelvis, abscesses of, 551
—, burrowing abscesses in, 510
—, crushing of acetabulum of, 669
—, exudation in, 431, 457
—, fibroma of, 511
—, fractures of, 668
— — pelvic fossa, 669
— — — ring, 668, 671
—, inflammatory process in, true, 286, 290
—, injuries of urethra in fractures, 504, 670
—, sarcoma of bones of, 511
— — muscles, 511
—, sinuses of, 451
—, true, cysts of, 431
—, tumours of, 511, 512
— —, examination of, 510, 511
— — in true, symptoms caused by compression and displacement, 431, 457, 509
Penis, constriction of, 505
—, deformities of, 505
Penis, injuries of, 505
—, osseous nodules in, 505
—, subcutaneous tumours of, 505
—, surgical diseases of, 505
—, ulcers of, 507
— —, cancerous, 507, 508
— —, tubercular, 507
Perichondritis, laryngeal, 122, 140
Pericolitis, 363
Perimetritis, intestinal obstruction in, 376
— with peritonitis, 289, 290
Perinephritis, 470
—, diagnosis from pleurisy, 470
— —, tubercular hip, 692
—, ileus in, 376
—, in renal tuberculosis, 491
—, origin of, 471
Periorchitis, 447
—, secondary, 447
—, serous and proliferating, 447
Periostitis of bones of skull, 55
— humerus, 610
— hyoid, 140
—, *see also* Osteomyelitis
— jaw, 63, 64, 83, 86, 90, 91, 140
— metacarpals, 643, 648
— tibia, 749
Periproctitis, 292, 452
Perisalpingitis, 289
Peritoneum, congenital pouches of, strangulation of bowel in, 389
—, inflammation of, 270-292
— —, blood condition in, 276, 277
—, tuberculosis of, 298
Peritonitis, 270-292
—, adhesive, 300, 302
—, commencing, 271
—, determination of leucocyte blood count in, 276, 277
—, physical condition in, 273
—, diagnosis from dysmenorrhœa, 279
— — gall-bladder pain, 279
— — hysteria, 278
— — ileus, 380
— — intestinal obstruction, 279, 280
— — mucous colic, 279
— — pneumonia and pleurisy, 276, 280
— — renal colic, 279
— — spinal caries, 280
— — tabes, 278
— — from strangulated hernia, 279
—, diffuse, 280
—, exudative, 300
— in a hernial sac, 425
—, indications for exploratory puncture in, 305

Peritonitis in intestinal injuries, 263
—, localized, 282
— —, in epigastrium, 282
— —, hypochondrium, 282
— —, hypogastric region, 283
— —, lumbar region, 282, 283
— —, true pelvis, 292
—, nodular, 301
—, origin of tubercular, 303
—, perforative, 272, 273, 282
—, peripheral (Lennander's), 330
—, prognosis of, 273
—, residual abscesses after, 272, 288, 355
—, sero-purulent, 272, 288, 355
—, serous, 301
— —, in appendicitis, 355
—, significance of age and sex in diagnosis of, 273
— — history in diagnosis of, 272, 273
—, tubercular, 264, 298, 299
— —, with saccular exudation, 300, 302, 307
Phantom hernia of lumbar region, 398, 399
— tumours of abdomen, 304
— of neck, 148
Pharynx, abscess of, 99, 104
—, adenoids in, 99
—, cancer of, 111
—, diphtheria in, 119
—, examination of, 119
— —, in dysphagia, 131
—, foreign bodies in, 131, 132
—, inflamed, diffuse and unilateral, 98
—, inflammatory processes in, 119
—, polypus of, 104, 105
—, pressure sore in, 111
—, sarcoma of, 104
—, syphilis of, 98, 107, 109, 111
—, teratoid tumours of, 105
—, ulcers in, and their diagnosis, 107, 109, 111
Phlegmon in axilla, 224
— ligneux, 144, 305
— of floor of mouth, 98
— front of arm, 603
— frontal sinus, 63
— mediastinum, 134, 141
— scalp, 55
— supraclavicular region, 141
Phosphorus necrosis of jaw, 89, 108
Pituitary gland and its relation to obesity, 185
— —, injuries of, 44
— —, tumours of, 25
Pleura, inflammation of, after peritonitis, 276, 280
Pleura, inflammation of, purulent, 222
—, injuries of, 202
— —, with cardiac wounds, 204
Pleural exudation in subphrenic abscess, 297
Pneumococcal peritonitis, 283, 302
Pneumonia, croupous, 208
—, diagnosis from diphtheria, 120
— — peritonitis in children, 280
—, metastatic, 276
—, purulent, 208
—, resulting in spinal caries, 555
—, traumatic, 125, 202
Pneumothorax, traumatic, 202
Pollakiuria, 460
Polyposis of rectum, 433
Polypus at neck of bladder, 492
— in nasopharynx, 104, 105, 131
— of ear, 28
Polyuria, 460
Pons, injuries of, focal symptoms, 47
—, tumours of, diagnostic signs, 26
Porencephaly, traumatic, 38, 58
Pott's deformity, 552
Pouches, ileo-appendicular and right retrocæcal hernia in, 390
Precentral sulcus, determination of, on skull, 51
Pressure diverticulum of œsophagus, 136
Processus vaginalis of peritoneum and herniæ, 410
Proctitis, gonorrhœal and syphilitic, 433
Prolapse of anus and rectum, 437
Prostate, abscess of, 458, 501
— —, diagnosis, 292, 466
—, carcinoma of, 500
—, cysts of, 512
—, examination of, 466, 467
—, gonorrhœa of, 501
—, hæmorrhage from, 500
—, hypertrophy of, 458, 498
— —, symptoms and course, 498, 499
—, sarcoma of, 500
—, tuberculosis of, 502
Prostatitis, 437
—, diagnosis of, 466
Pseudo-hermaphroditism, 406, 407
Pseudo-leukæmia, glandular enlargement in, 165, 216
—, pharyngeal swelling in, 103
—, splenic enlargement in, 352
Psoas abscess, 471, 551
Pupillary reaction, hemianopic, 23
Pupils in cerebral pressure, 13
—, reaction of in vertebral injuries, 529
— — in visual disturbances in, 42

Pyelitis, 478, 479, 480
—, anatomical diagnosis of, 480
—, gonorrhœal, 480
— in prostatic hypertrophy, 498
— of pregnancy, 480
Pyelonephritis, 479
Pyloric cancer, 328
— —, signs of retention in, 328
— —, skiagrams of, 317
— spasm, 323
— stenosis, cancerous, 310, 320, 324
— —, innocent, 324
— — of infants, 324
— ulcer, 323
Pyonephrosis, 476
Pyorrhœa alveolaris, 108
Pyosalpinx, 289, 290

Q

QUADRICEPS tendon, laceration of, 712

R

RACHISCHISIS, posterior, 514
Radial paralysis, posture of hand in, 631
Radio-carpal joint, dislocation of, 621
Radius, absence of, 632
— fracture, 612-617
— —, combined, 615, 617
— —, greenstick, 613
— — —, with fork-like posture, 613
— — — —, into joint, 613
—, head of, chisel fracture, 599, 601
—, individual dislocation of, 601
—, separation of epiphysis, 614, 615
Ranula, 100
—, differential diagnosis, 100, 101, 102
Raynaud's disease, 644, 646,
Recklinghausen's disease, 60, 180
Reclus's disease, 238
Rectal fistulæ, 435, 436, 451-454
— —, acquired, inflamed, 452
— —, complete and incomplete, 453
— —, congenital, 451
— —, ischio-rectal and pelvi-rectal, 453
Rectum, carcinoma of, 432, 433, 435
— —, causing perirectal suppuration, 292
— — — stenosis, 373
—, condition of, in spinal injuries, 492
—, fissure of, 434
—, fistulæ of, 436, 451-454
—, foreign bodies in, 437
— — —, introduced into, 438
—, gonorrhœal stricture of, 432
Rectum, injuries of, 438, 439
—, polyposis of, 433
—, prolapse of, 438
—, sarcoma of, 434, 436
—, syphilis of, 362, 374, 432, 433
—, tenesmus of, 431
—, tuberculosis of, 362, 432
Recurrent nerve paralysis, 213
— — —, symptoms of, 127
Reflex epilepsy, 39
— symptoms after wounds of heart, 205
— — of chronic purulent otitis, 31
Resin stone in stomach, 320
Retention swellings of kidney, 487
Retrobulbar fatty tissue, venous thrombosis of, 54, 65
Retroflexion of gravid uterus causing constipation, 431
— — — causing ileus, 375
Retropharyngeal abscess, 99, 121
— —, important diagnostic signs of, 131
Retroperitoneal inflammation, 282
Retrotonsillar abscess, 99
Rhagades on tongue, nature of, 115
Rheumatism of ankle, 772, 773
— elbow, 607
— hip, 680, 691, 694
— knee, 721, 722, 723
— —, ankylosing, 728
— shoulder, 584, 585
— wrist, 624
— —, chronic, 625, 626
Rhinoscleroma, 111
Ribs, chondritis of, after typhoid, 227
—, fractures of, 200
—, gumma of, 227
—, ostitis of, 227
—, protuberance of, 562, 563, 566
—, tuberculosis of, 227, 236
Rickets, bone changes in, 628, 676
—, protuberance on ribs, due to, 566
Rider's bone, 699
Risus sardonicus in tetanus, 83
Rodent ulcer of face, 73, 74, 75, 76
Roser-Nélaton's line, determination of, 652
— — — in congenital dislocation of hip, 672
Rotation dislocation of cervical vertebra, 194
— fracture of thigh, 664, 665, 666

S

SACRAL tuberculosis, 549, 550
Sacro-lumbar tumours, congenital, 517

Salivary glands, swelling of, 97
— — —, diagnosis from tumours of neck, 149
— stones in Steno's duct, causing acute swelling of parotid, 67
— — Wharton's duct, causing inflammatory swelling of submaxillary, 140
Salpingitis, 285
—, diagnosis from appendicitis, 290
Sarcoma of abdominal wall, 397
— arm, 607, 611
— back of neck, 185
— bladder, 497
— cervical glands, 181
— clavicle, 226
— diploe, 61
— femur, 698, 699, 700, 702
— gluteal region, 731
— gum, 102
— hand, 639
— kidneys, 486, 487
— knee-joint, 726
— lower jaw, 95, 96
— lung, 221
— mamma, 247
— mediastinum, 221
— neck, 180
— —, pulsating, 173
— nose, 106
— os calcis, 773
— parotid gland, 179, 180
— patella, 729
— pelvis, 511, 731
— penis, 505
— pharynx, 104
— prostate, 500
— rectum, 434, 436
— sacral region, 517
— scapula, 227
— scrotum, 440
— shoulder, 587
— skull, 61
— spermatic cord, 442
— spinal cord, 543
— spleen, 352
— submaxillary gland, 179
— testicle, 449
— thigh, 699, 700, 702
— thorax, 229, 232
— thyroid, 163
— tibia, 699, 745, 746, 748
— tongue, 114
— tonsillar region, 103
— trachea, 129
— umbilicus, 397
— upper jaw, 91, 92
— vascular sheaths of neck, 181
— vertebral column, 542
Scalp, fibroma of, 59
—, gumma of, 60, 62
—, sebaceous cysts of, 59, 60, 62
Scaphoid of foot, fracture of, 768
— hand, fracture of, 619, 620
Scapula, fracture of, 577, 580
—, osteo-myelitis of, 224
—, sarcoma of, 232
—, tuberculosis of, 229
Scarlatinal arthritis of hip, 684, 685
— — shoulder, 584
School scoliosis, 566
Sciatica, 731-734
—, diagnosis from hip disease, 692
—, scoliotic, 556, 557, 734
Scirrhus of breast, 239, 241, 245
Sclerosis, multiple, paraplegia in, 542
Scoliosis, 559-567
—, causes of, 565
—, complete, 564
—, fixed, 564
— ischiadica, 556, 557, 734
—, mobile, 564
— of dorsal vertebræ, primary, 563, 565
Scrofula, tubercular lymphoma, 164, 165
Scrotum, acute inflammation of, 439
—, carcinoma of, 441
—, contents, 441
—, dermoid of, 440
—, elephantiasis of, 440
—, fibroma of, 440
—, lymphangioma of, 440
—, sarcoma of, 440
—, sebaceous cyst of, 440
—, swellings of, 440
—, tumours of, 440
—, urinary infiltration of, 439
Scurvy, gangrene in, 99
—, hæmorrhage from gums in, 108
Sebaceous cyst of face, 67, 68
— — hand, 638
— — neck, 175, 184
— — penis, 505
— — root of nose, 68
— — scalp, 59, 60
— — —, malignant degeneration of, 62
— — scrotum, 440
— — skin of back, 229
— — skull, 59
— — — with sinus formation, 59
— — umbilicus, 402
Segmental diagnosis of spinal cord, 526-531, 543
Semilunar bone, dislocation of, 618, 619
— —, fracture of, 620, 621

Semilunar cartilage, incarceration of, 711
— —, laceration and displacement of, 711
Seminal vesicles, abscess of, 458
Sensation in vertebral injuries, 522, 523
— — —, partial, 525
Sesamoid in knee-joint, 710, 711
— of tarsal bones, fracture of, 770, 771
Shoulder, rigidity of, 582
Shoulder-joint, contusion of, 580
—, dislocation of, 573, 574, 578
—, elevated level of, congenital, 558
—, fractures of, 574, 575, 576, 577, 578, 579
—, gonorrhœa of, 585
—, injuries of (examination for), 572
—, inflammatory processes, 581
— — —, diagnosis from diseases of bursa and of joint, 581, 582
—, primary disease of bones of, 587
—, sprain of, 580
—, testing movements of, 582, 583
—, tubercle of, 584, 585
Sigmoid, cancer of, 373
—, stenosis of, 379
—, volvulus of, 388
Sigmoiditis, 292
—, ulcerating, 363
Sinus at umbilicus, 402
—, cavernous, thrombosis of, 65
— from biliary tract, 402
— intestine, 402
— rectum, 451, 452
— stomach, 402
— thyroid gland, 147
— urachus, 402
— vitelline duct, 401
— frontal phlegmon, arising therefrom, 65
— in perinæum, 451, 452
—, longitudinal, angioma of, 57
—, maxillary, chronic inflammation of, 90
— of abdomen, 401, 402
— branchial cleft, 146
— inguinal region, 402
— urinary tract, 402, 454
— on neck, 145
— —, actinomycotic, 145
— — (complete and incomplete), 146
— —, congenital, 146, 147
— —, gummatous, 145
— —, traumatic, 145
— —, tubercular, 145
—, pericranial, blood cyst of, 53
Sinus, secretions of, 402
— thrombosis, after aural suppuration, 32, 35
Sinusitis, acute, 64
——, chronic, 64
—, frontal, 65
Situs inversus of unpaired abdominal viscera, 252
— —, partial inferior, 252
Skin appendages in front of ear and on face, 68
— cancer of, on foot, 789, 791
— —, on face, 72-76
— —, on hand, 639
— —, on umbilical region, 397
— reflexes in spinal injuries, 524
—, seborrhœa of, 62
— —, on forehead and temples, 79
Skull, acute inflammatory processes on, 55
—, base of, fibroma of, 105
— —, fractures of, 3, 6
— —, nerves at, 49
— —, sarcoma of, 104
—, chronic inflammatory swellings of, 60
— —, ulcerating growths of, 62
—, demarcation of cortical centres on the surface of, 46, 47, 51
—, dermoid of, 57, 58
—, erysipelas of, 55
—, exploration of, 17
—, fissures of, 6
—, fractures of, 1
—, contrecoup fractures of, 7
— —, direct symptoms, 2
— —, indirect symptoms, 4
— —, position and course of, 5, 6
— —, X-ray diagnosis of, 7
—, gummata of, 60, 62
—, injuries of, causing epilepsy, 37, 38
—, new growths of internal layer, 23
—, osteoma of, 59
—, osteomyelitis of, 55
—, periostitis of, 55
—, phlegmon of glands of, 55
—, sarcoma of, 60, 61
— —, periosteal, 61
—, sebaceous cyst of, 59
—, secondary growths of, 62
—, tubercle of, 60, 61
Small intestine, gall stones in, 292
— —, intussusception of, confusion with appendicitis, 292
— —, palpation of, 256
— —, stenosis of, 371, 377, 382
— — — through fibro-sarcoma, 369

Small intestine, torsion of, 388
— —, tumours of, 306
Solitary kidney, displacement of, 251
Spermatic cord, hydrocele of, 410, 411, 441
— — —, diagnosis from inguinal hernia, 412
— —, tumours of, 441
Spermatocele, 446
Sphincter of bladder, ulceration of, as a cause of incontinence, 460
Spina bifida, 232, 513
— —, forms of, 513
— — occulta, 517
— — —, club foot in, 78
— — —, pes cavus in, 783
Spinal caries, 544-553
— —, ankylosing, 554
— — causing bending of spine, 552
— —, cervical, 547, 549, 550
— — —, commencing, 546
— — commencing, 545
— —, diagnosis from peritonitis, 263
— — — spinal cord tumour, 541
— — — sarcoma of vertebra, 542
— —, non-tubercular, 553
— — —, ankylosing, 554
— — —, secondary after pneumonia and typhoid, 555
— — of dorsal vertebræ, 549, 551
— — lumbar vertebræ, 547, 549
— —, osteo-myelitic, 555
— —, posture of head in, 192, 548
— —, syphilitic, 555
— —, traumatic, 554
— —, tubercular, 192, 229, 544-553
— — —, in adults, 546
— — —, in children, 544, 545
— — with burrowing abscess, 549
— — cord symptoms, 553
— — without protuberance and without burrowing abscess, 544
— cord, Brown-Sequard's syndrome, 525, 541
— —, compression of, 537, 553
— —, congenital clefts in, 485
— —, contusion of, 525
— —, gumma of, 542
— —, hæmorrhage in, 526
— —, injuries of, 520
— — —, complete, 525
— — —, partial, 525
— —, non-traumatic diseases of, 540
— —, sarcoma of, 542
— —, segmental diagnosis, 526, 527, 528, 529
— —, solid tubercle of, 542
— —, tumours of, 540, 541
Spinal meninges, innocent growths of, 543
Spine of tibia, detachment of, 712
— — —, normal picture of, 712
Spleen, abscess of, 282, 351
—, amyloid disease of, 352
—, congenital syphilis of, 352
—, displacement of, 254
—, hypertrophy of, 351, 352
—, injuries of, 263
—, swelling of, in diphtheria, 120
—, tumours of, 352
Sporotrichosis of elbow region, 609
Staphylomycosis of breast, 236
— medulla of femur, 705, 706
— tibia, 743
Sterno-clavicular joint, injuries of, 569
Sterno-mastoid, gumma of, 149
— —, myositis of, 141
— —, tuberculosis of, 149
Sternum, abscess of, 225
—, fracture of, 200, 201
— —, transverse, in upper portion, 537
—, gumma of, 227
—, malignant growth of, 227
—, osteo-myelitis of, 141
—, tubercle of, 226, 227
Stomach, abnormalities in position of, 254, 255, 320
—, cancer of, 325-329
— — at cardia, 327
— — fundus, 327
— — pylorus, 328
— —, chemistry in, 314, 315, 327
— —, varieties of, 327, 328
—, cicatricial stenosis of, 316, 320
—, contusion of, 261, 262
—, dilatation of, 320
—, foreign bodies in, 320
—, gunshot wounds of, 268, 269
—, hæmorrhage from, 312
— hour-glass, 322, 323
—, operative fixation of, 321
—, perforation of, 329
— —, differential diagnosis, 331
— —, local symptoms, 330
—, shape and position of, abnormal, 320, 321
— — —, normal, 256
—, sinuses from, 402
—, skiagram of, 316, 317, 318, 319
— — in retention of contents, 319
— —, technique and diagnostic significance, 315
—, surgical diseases of, 309
— — —, with peritoneal signs, 282
—, ulcer of, 321

Stomach, ulcer of, diagnosis from cholecystitis, 335, 336
— — — ileus, 381
— — prolonged course of, 322
— —, perforating, 282, 329, 330
— —, uncomplicated, 321, 322, 323
Stomatitis, gangrenous, 84, 99
—, mercurial, 84
Stones of kidney, infected, 483
— —, non-infected, 482
Stools, blood in, 261, 371, 387, 431
—, diagnostic significance of their consistence, 432
—, incontinence of, 524
—, pus in, 371
—, retention of, 370
—, shape of, 371, 372, 432
Streptococcal laryngitis, diagnosis from diphtheria, 119
Streptomycosis of breast, 236
Stricture of rectum, gonorrhœal and syphilitic, 433
—, urethral, examination of, 467
Styloid process of radius, fracture of, 613, 614
— — ulna, fracture of, 613, 617
Subluxation of foot, 760, 761
— radius, 597
Submaxillary gland, inflammation, 140
— — —, chronic, 176
— —, mixed tumours of, 177
— —, sarcoma of, 179
— —, tubercle of, 177
Suppuration after gunshot wounds, 804
Surgical emphysema after injuries to air passages, 123
— —, lung injuries, 201, 202
Swallowing, difficulty in, 130
— —, position and nature of obstruction, 135
— —, through affections of mouth and throat, 130-131
— — — foreign bodies, 131
— — — laryngeal diseases, 125
— — — œsophageal disease, 132
Sweat glands of axilla, inflammation, 224
Synovitis of ankle, tubercular, 773, 774
— knee, osteo-myelitic, 723
— —, rheumatic, 723
— —, syphilitic, 723
Syphilis insontium, 23, 98
— of brain, 23
— elbow, 604
— epididymis, 442, 454
— face, 69, 70
— finger, 641, 644
— foot, 791
Syphilis of hand, 641, 644
— intestine, 374
— knee, 723
— large intestine, 362
— larynx, 125, 126, 127
— leg, 735, 737, 738
— lips, 69, 108
— mouth, 98, 108, 109
— neck, 145, 165
— nose, 71, 72
— œsophagus, 137
— palate, 103
— penis, 507, 508
— rectum, 362, 433
— testicle, 449
— thoracic wall, 225
— thyroid, 162
— tibia, 746, 748, 749
— vertebræ, 555
Syringo-myelia, hand deformity in, 645, 646

T

Tabes, diagnosis from perforation of stomach, 331
— — peritonitis, 278, 279
— — tumour of spinal cord, 541
Talipes, 776-785
Tar cancer, 441
Tear ducts, inflammation of, 64, 65, 176
Tegmen tympani, suffusion of, 7
Temporal lobe, abscess of, after otorrhœa, 31
— —, tumours of, 25
Tendon reflexes, condition of, in spinal caries, 553
— — — vertebral injuries, 524
Tenesmus of bladder, 456, 488, 496
— — in stone, 491
— — urinary tubercle, 488
— rectum, 431
— — with constipation, 432
Teno-synovitis of hand, 622, 646
— —, gonorrhœal, 647
—, tubercular, 625, 626
Teratoma of pharynx, 105, 106
— sacral region, 517
Testicle, gumma of, 449
—, hæmatocele of, 443, 447
—, hydrocele of, 412, 413, 447
—, infarction of, 445
—, inflammation of, 442, 443
— —, metastatic, 444
—, injuries of, 443
—, spermatocele of, 446, 447
—, strangulation of, 423

Testicle, syphilis of, 449
—, torsion of, 423, 445
—, tumours of, 448, 449
Tetanus of head, with facial palsy, 83, 84
— after wounds of warfare, 806
Thigh, abscess of, 552
—, aneurism of, 698, 699
—, blood cysts of, 699
—, dislocations of, 655-661
— —, congenital, 671
—, fractures of, 661-668
—, muscular swelling of, 699
—, osteo-myelitis of, 697
—, tumours of, 696-700
— —, in epiphysis, 701
— — diaphysis, 705
Thorax, angioma of, 229
—, bones of, fracture, 199-201
—, burrowing abscess of, 550
—, empyema, breaking through, 223
—, enchondroma of, 232
—, fibroma of, 230
—, inflammatory processes of, 225-229
—, injuries of, 199, 200
—, lipoma of, 229-231, 550
—, lymphangioma of, 230
—, malignant tumours of, 229, 232, 233
— muscle, gumma of, 225
—, osteoma of, 232
—, osteo-myelitis of, 224
—, sarcoma of, 227, 228, 229
—, sebaceous cyst of, 229
—, surgical diseases of, 199
— — —, external, 223
— — —, internal, 212
—, tuberculosis of, 223, 225
—, tumours within, 212
— — without, 223
Thrombo-phlebitis of transverse sinus in aural suppuration, 32, 35
Thymus, condition of, in Graves's disease, 154
—, hypertrophy of: diagnosis, 214
Thyroid, cancer: secondary deposits, 162, 163, 164
— — — —, symptoms, 162
— fistulæ, 147
— gland in obesity, 185
— —, inflammation of, 141, 160
— —, simple hyperplasia of, 149, 150
— —, tumours independent of, in neck, 164
Tibia, chronic abscess of, 746, 750
—, dislocation of, 718, 759
— —, posterior, 728, 729
— —, at lower end, 759, 760, 761
Tibia, fractures of, 717
—, laceration of crucial ligaments, 711
—, normal, 749
—, osteo-myelitis of, 743, 746, 747, 748, 749
—, periostitis of, 748, 749
— —, syphilitic, 748
—, syphilis of, 746, 749
—, tuberculosis of, 743, 748, 750
—, tumours of, 743, 744, 745, 746
Tic, rotatory, 198
Toe-nail, ingrowing, 791
Toes, chondroma of, 787
—, deformities of, 787
—, gangrene of, 776
Tongue, actinomycosis of, 114, 115
—, angioma of, 113
—, cancer of, 107, 114, 115, 116
—, dry (significance of), 112
—, fibroma of, 114
—, gumma of, 114, 115, 116
—, leucoplakia of, 112
—, lipoma of, 113, 114
—, lymphangioma of, 113
—, primary chancre of, 115
—, sarcoma of, 114
—, swelling of, 98, 113
—, tuberculosis of, 114, 115
Tonsil, cancer of, 111
— —, metastasis in neck, 170
—, gumma of, 109
—, mixed tumours of, 103
—, primary chancre of, 110
—, sarcoma of, 103
—, tuberculosis of, 109
Tonsillitis, 119
Torticollis, 192, 196, 197, 198
Trachea, surgical diseases of, acute, 118
— — —, chronic, 125
Trismus, 83
Trochanter, fractures of, 662, 663, 664, 665
—, level of, 653, 673
— —, in congenital dislocation, 673, 677
— —, coxa adducta, 680, 681
Tubal pregnancy, ruptured, 285, 289, 290
— — —, diagnosis from gastric ulcer, 331
— — — — ileus, 381
Tubercle of abdominal wall, 399
— arm, 610, 611, 612
— bladder, 490, 495
— brain, 19, 23
— cervical vertebræ, 193, 544-553

Tubercle of elbow, 604, 605, 606
— epididymis, 446
— femur, 686-691
— finger, 644, 647, 648
— floor of mouth, 107, 109
— foot, 773, 774, 791
— gum, 84, 109
— hand, 637, 638
— hernial sac, 426
— hip, 686
— humerus, 585, 586, 610, 611
— inguinal glands, 398
— intestine, 303
— jaw, 65, 84, 87, 88, 89
— kidney, 472, 478, 488
— knee, 723-729
— lachrymal sac, 65
— larynx, 126, 127
— leg, 748
— lobule of ear, 78
— lungs, suppurative, 211, 223
— mamma, 236, 237
— neck, 145, 149, 165
— nose, 111
— os calcis, 773
— palate, 111
— parotid, 178, 179
— pelvic bones, 692
— peritoneum, 280, 298
— pre-patellar bursa, 729
— prostate, 502
— rectum, 362-433
— ribs, 227
— scapula, 229, 585
— shoulder, 584, 585
— skull, 60, 61
— sternum, 226, 227
— subdeltoid, 584
— submaxillary gland, 177
— tendon sheaths of hand, 625, 626
— thorax, 223, 225
— thyroid, 162
— tibia, 743
— —, at lower end, 748, 750
— tongue, 114, 115
— tonsillar region, diagnosis from gumma, 109
— urinary passages, 488
— vertebræ, 192, 228, 544, 548
— wrist, 625, 626, 627
Typhoid fever, arthritis of hip, after, 695
— — — vertebral joints, after, 554
— —, ostitis and chondritis of ribs, after, 227
Typhlitis, ulcerative, 363

U

ULCERS of concha of ear, 79
— of duodenum, 323
— face, 69-80
— foot, 789, 790
— gum, 108
— intestine, 374, 381
— larynx, 122, 127
— leg, 734, 735, 736, 737
— oral cavity, 107
— penis, 507, 508
— scalp, 62
— scrotum, 440
— stomach, 321
— tongue, 115
— tubercular, of gum, 87
Ulnar paralysis, posture of hand and fingers in, 629, 634
Umbilical cord, hernia of, 394, 396
— hernia, 394
— —, in cirrhosis of liver, 396
— —, strangulation of, 428
Umbilicus, abscess of, 401
—, concretions in, 402
—, fistulæ of, congenital and acquired, 401
—, tumours of, 395, 402
Urachus, cysts of, 397
—, fistulæ of, 401
Ureter, catheterism of, 455, 469, 489
—, in urinary tuberculosis, 489
Ureteral stones, 483
Urethra, fistulæ in, 454
—, foreign bodies in, 458
—, hæmorrhage from, 463
—, injuries of, 502, 503
— —, in fractured pelvis, 504, 670
—, obstruction of, 457, 458
—, stricture of, 467, 468
— —, of gradual onset, 459
Urethritis, differential diagnosis in, 456
Urethroscopy, 467
Urinary disturbances, 456, 468
— —, difficult and painful micturition, 456, 457
— —, involuntary micturition, 456
—, fever, 481, 484
—, fistulæ, 402, 454, 455
—, gravel, 464, 482
—, tract, surgical diseases of, 455
— — — —, local symptoms, 465
— —, tuberculosis of, 463, 464, 488
Urine, albumin in, 462, 464
—, blood in, 265, 267, 464, 478, 485, 490, 496
—, examination of, 462, 463, 465

Urine, extravasation of, 439
— —, in injury of kidneys, 265
— —, rupture of bladder, 266, 267
— —, urethral injuries, 503
—, pus in, 461, 462, 479
—, retention of, 456
— —, results, 476, 487
—, sugar in, in pancreatic disease, 351
Urticaria, in hydatid disease, 347
—, hydronephrosis, 476
Uterus, cancer of, causing intestinal obstruction, 376
—, cystoma of, 307
—, fibrous myoma of, 306
—, inflammation in and around, 285
—, pregnant diagnosis from abdominal tumour, 307

V

VARICOCELE, 411
—, with atrophy of testicle, 413
Varicose veins of leg, 733, 737
— — thigh, 698
Varix, aneurismal, diagnosis from arterio-venous aneurism, 174
Vein, internal jugular, thrombophlebitis of, 35
—, saphenous, dilatations of, 416
Verruca senilis, 62
Vertebral column, arthritis deformans of, 554
— —, congenital deformities of, 513, 558
— —, curvatures of, 555-567
— —, dislocations of, 188, 538, 539, 540
— —, fractures of, 188, 520, 535-538
— —, injuries of, 520-540
— —, osteo-myelitis of, 192, 555
— —, partial injuries of, 525
— —, rigidity of, 554
— —, sarcoma of, 543
— —, sprains of, 519
— —, syphilis, tertiary of, 555
— —, tubercular abscess of, 143, 148, 228, 548, 549, 550
— —, tumours of, 540, 541
— — —, diagnosis from spinal caries, 541
Vertebral injuries, examination of, 521
— — — for movements, 521
— — — reflexes, 524
— — — sensation, 522, 523
— — — vasomotor state, 523
— — — visceral functions, 523
Vestibular nerve, disturbances of, 48
Volvulus, 388
—, ileo-cæcal, 388
— of sigmoid flexure, 388
Vomiting, varieties of, 311, 312
— of stomach, 321

W

WARTS, soft, of face, 68
Whitlow, 641, 642, 643
Wrist, abnormal postures of, 632
—, arthritis of, 623, 624, 626
—, bones of, fracture of, 615-621
—, dislocation, fracture of, 619, 620
— — of, 618, 621
—, gonorrhœa of, 624
—, injuries of, 612
— —, scheme, 622
—, normal, 618
—, rheumatism of, 624
— —, chronic, 625
—, sprain of, 612, 613, 622
—, tuberculosis of, 625, 626, 627
Wry-neck, 192, 196, 197, 198

X

XANTHELASMA of face, 68
X-ray, dermatitis of hand, 639
— diagnosis in diseases of stomach, 315
— — of foreign bodies in war wounds, 796

Y

Y-SHAPED fracture of femoral condyles, 715, 717
— — head of humerus, 576
— — ligament in hip dislocation, 657
— — lower end of humerus, 590, 598, 603
— — neck of femur, 663

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